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DISEASES OF THE LIVER

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CLINICAL LECTURES

ON

DISEASES OF THE LIVER

JAUNDICE AND ABDOMINAL DROPSY

INCLUDING THE CROONIAN LECTURES ON
FUNCTIONAL DERANGEMENTS OF THE LIVER DELIVERED AT THE
ROYAL COLLEGE OF PHYSICIANS IN 1874

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BY

CHARLES MURCHISON, M.D. LL.D. F.R.S.

FELLOW OF THE ROYAL COLLEGE OF PHYSICIANS; PRESIDENT OF THE PATHOLOGICAL SOCIETY OF
LONDON; PHYSICIAN AND LECTURER ON THE PRINCIPLES AND PRACTICE OF MEDICINE,
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HOSPITAL, AND ON MEDICAL STAFF OF H.M. BENGAL ARMY

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TO

WILLIAM MURRAY DOBIE, M.D.

PHYSICIAN TO THE CHESTER INFIRMARY

THIS WORK IS DEDICATED
BY
THE AUTHOR

IN ADMIRATION OF HIS TALENTS AS A SCIENTIFIC PHYSICIAN

AND IN TOKEN OF A FRIENDSHIP OF THIRTY YEARS

PREFACE

TO

THE SECOND EDITION.

As was stated in the first edition, these Lectures have no pretension to be a systematic treatise on Diseases of the Liver. Their sole object is to assist the student and practitioner in the diagnosis and treatment of these maladies.

The favourable reception accorded to the first edition encourages me to hope that the work answered the purpose for which it was intended. Five years have now elapsed since the last copy of a large impression was disposed of.

The delay in the preparation of this edition has been occasioned by other avocations of a literary and professional character, and by my desire to include the results of the labours of my contemporaries, as well as those of my matured experience derived from hospital and private practice. The Lectures have been in great measure re-written. Of the 96 cases which were published in the first edition 6 have been omitted, and in this edition 90 cases appear for the first time, making a total of 180. Most of these additional cases have been the subject of clinical remarks, which have been incorporated with the original Lectures. The woodcuts have been increased from 25 to 37.

published in the 'Edinburgh Medical Journal' for December 1865; and to the last Lecture have been added the results of an inquiry into the pathological consequences of gall-stones, commenced many years ago, and part of which appeared in a memoir on abdominal fistulæ, published in the 'Edinburgh Medical Journal' for July and August 1857. To all of the Lectures has been appended a history not only of those cases on which each Lecture was originally founded, but of others which have occurred subsequently and been the subject of clinical remarks in the wards. These histories have been condensed from notes taken at my dictation by my clinical clerks, whose kind and ready assistance I take this opportunity of acknowledging. The records of these cases will, it is believed, be useful to the medical practitioner who meets with others of a like nature, for, as the founder of pathological anatomy long ago observed: 'Nulla est alia pro certo noscendi via, nisi quamplurimas et morborum et dissectionum historias, tum aliorum, tum proprias, collectas habere, et inter se comparare.'—Morgagni, de Sed. et Causis Morbor. Lib. IV. Prooemium.

79 Wimpole Street, Cavendish Square, W.
June 1868.

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FIG.
22. M
23. A
24. M

25. M
26. M
27. Sh

28. M

29. Mi

30. Pe

31. Pe

32. Cr
33. Gl
34. Cr
35. La
36. Cr
37. Gl

TABLE OF CHEMICAL EQUIVALENTS.

Albuminoids (Lieberkühn)	$C_{72}H_{112}N_{18}SO_{23}$
Excretin	$C_{78}H_{156}SO_2$
Taurocholic acid	$C_{26}H_{45}NO_7S$
Glycocholic acid	$C_{26}H_{43}NO_6$
Cholic acid	$C_{24}H_{40}O_5$
Taurin	$C_2H_7NO_3S$
Glycocin	$C_2H_5NO_2$
Bilirubin	$C_{16}H_{18}N_2O_3$
Biliverdin	$C_{16}H_{20}N_2O_5$
Tyrosin	$C_9H_{11}NO_3$
Leucin	$C_6H_{13}NO_2$
Hippuric acid	$C_9H_9NO_3$
Xanthin	$C_5H_4N_4O_2$
Cystin	$C_3H_7NSO_2$
Kreatin	$C_4H_9N_3O_2$
Kreatinin	$C_4H_7N_3O$
Uric or lithic acid	$C_5H_4N_4O_3$
Urea	CH_4N_2O
Oxalic acid	$C_2H_2O_4$
Starch	$C_6H_{10}O_5$
Dextrin	$C_6H_{10}O_5$
Glycogen, or animal starch	$C_6H_{10}O_5$
Cane-sugar	$C_{12}H_{22}O_{11}$
Glucose, or grape-sugar	$C_6H_{12}O_6$
Lactose, or milk-sugar	$C_6H_{12}O_6$
Inosite, or muscle-sugar	$C_6H_{12}O_6$
Lævulose	$C_6H_{12}O_6$
Cholesterin	$C_{26}H_{44}O$

Corrigenda.

Page 60, line 28, *for* Case XCVI. *read* Case XCII.
,, 142, ,, 12, *for* Case CLXXVI. *read* Case CLXXVIII.
,, 156, ,, 8, *omit* See Case CXXI.
,, 183, ,, 18, *for* usually *substitute* occasionally.
,, 260, ,, 10, *insert at end of paragraph* See Lect. XI. p. 407.

LECTURE I.

ENLARGEMENTS OF THE LIVER.

INTRODUCTORY REMARKS—NORMAL DIMENSIONS AND BOUNDARIES OF THE LIVER—CIRCUMSTANCES UNDER WHICH ENLARGEMENT OF THE LIVER IS SIMULATED, AND THE MEANS OF DISTINGUISHING SUCH SPURIOUS ENLARGEMENTS: 1. CONGENITAL MALFORMATIONS; 2. EARLY LIFE; 3. RICKETS; 4. TIGHT-LACING; 5. CERTAIN DISEASES OF THE CHEST; 6. TUMOUR BETWEEN THE LIVER AND DIAPHRAGM; 7. ABNORMAL CONDITIONS OF THE ABDOMINAL VISCERA; 8. ABNORMAL CONDITIONS OF THE ABDOMINAL PARIETES—CASES IN ILLUSTRATION.

GENTLEMEN,—In systematic lectures on Medicine, it is the custom to describe in detail the numerous symptoms which characterise different disorders. It requires, however, little experience to discover that there are symptoms and signs which are common to many diseases, and that no small difficulty is often encountered in determining to which of its many sources a particular symptom ought to be referred. Yet this determination must always be your first object in practice. You must never rest satisfied with treating merely a symptom without endeavouring to acquire some definite notion of the local or general disease upon which it depends. In all cases of disease presenting some prominent symptom, you ought to ask yourselves two questions: 1. What are the different causes which may give rise to the symptom in question? and 2. Which is the most probable cause in the individual case before you? Not until you have given a satisfactory reply to these enquiries will you be in a position to speak with any confidence as to prognosis, or to adopt a rational method of treatment.

To no class of maladies are these remarks more applicable than to diseases of the liver. There are few diseases more difficult to discriminate, and perhaps none in which an erroneous diagnosis is oftener made: while symptoms depending upon disease of the stomach, the intestines, or the kidneys, or even of the heart, the lungs, or the brain, are constantly ascribed to derangements of the liver. It will be my object in these lectures to point out to you the chief signs and symptoms resulting from hepatic disease, the different morbid conditions from which each of them may arise, the rules by which you must be mainly guided in determining the precise disease in each case, and the conclusions to which you ought in this way to be led respecting prognosis and treatment. We shall commence, for instance, by discussing the different causes of Enlargement of the Liver; and in subsequent lectures, the causes of Atrophy of the Liver, of Jaundice, Hepatic Pain, Hepatic Dropsy, &c., will be duly considered.

ENLARGEMENTS OF THE LIVER.

Before proceeding to consider the various causes of true enlargement of the liver, it is necessary to have an accurate knowledge of its normal dimensions and boundaries, and also to keep in view certain conditions which during life may simulate enlargement.

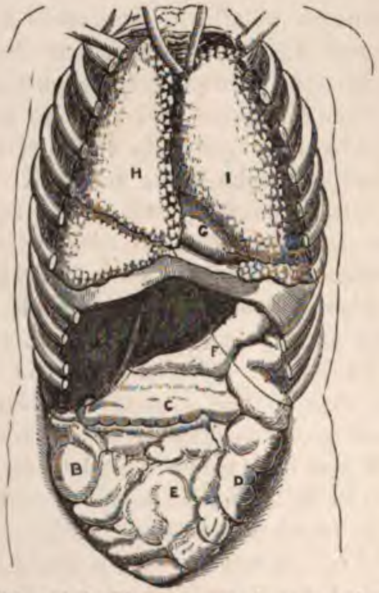

Fig. 1. Natural Position of the Liver, as seen after removal of the anterior wall of the chest and abdomen. Modified from Sibson's Med. Anatomy.

A, Liver. B, Ascending colon. C, Transverse colon. D, Descending colon. E, Small intestines. F, Stomach. G, Heart. H, Right lung. I, Left lung.

Normal situation and dimensions of the liver.—The liver is situated in the right hypochondrium, the convexity of the right lobe corresponding to the concavity at the base of the right lung with the diaphragm interposed, and the under surface being opposed to the stomach and large intestine, the right kidney and supra-renal capsule. The convex upper surface projects up into the right side of the chest, and a great part of it is in immediate juxtaposition with the ribs, but the uppermost portion (in a vertical direction) is separated from the wall of the chest by the thin lower margin of the right lung. (See fig. 1.) Accordingly, in percussion during life, the upper

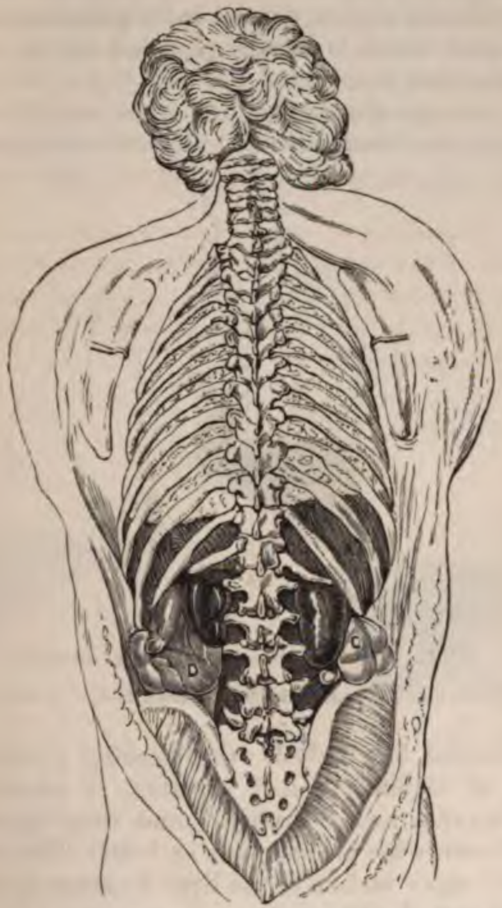

Fig. 2. Natural Position of the Liver, as seen after the removal of the posterior wall of the chest and abdomen. Modified from Sibson's Med. Anatomy.

The liver is covered by the diaphragm, beneath which, on the left side (B) there is also the spleen and a portion of the stomach. A, Right lobe of liver. C, Ascending colon. D, Descending colon.

margin of hepatic dulness may be said to be twofold, one boundary limiting the region where the organ is in close approximation to the walls of the chest, and where the dulness is absolute, the other corresponding to the extreme height of the liver, and including the space where it is overlapped by the thin layer of lung, and where the sound on percussion constitutes a transition from the hepatic dulness to the pulmonary resonance. It is the latter which is usually regarded as the true upper margin of the liver (fig. 3).

There is a peculiarity in the upper margin of hepatic dulness which is of some practical importance—namely, that it is not horizontal, but arched. Commencing posteriorly about the tenth or eleventh dorsal vertebra, it ascends slightly towards the axilla and the nipple, and then again descends gradually towards the median line in front. The arched character of the upper surface of the liver is shown in the annexed diagrams (figs. 3, 4, 5).

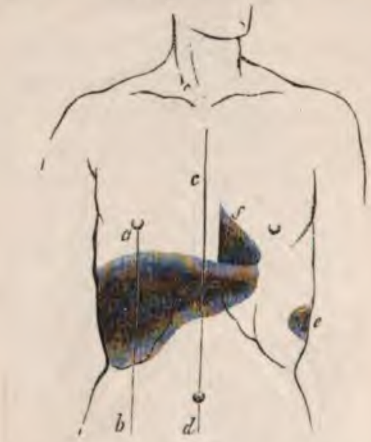

Fig. 3. Area of Hepatic Dulness, viewed anteriorly.

a-b, Right mammary line. *c-d*, Median line. *e*, Splenic dulness. *f*, Cardiac dulness.

In determining the upper margin of hepatic dulness we must trust to percussion alone. In ordinary cases it is sufficient to note the upper limit in what is called the *right mammary line*, or a line descending perpendicularly from the right nipple (fig. 3). Here, in a healthy adult, the *true* upper margin of the liver is situated in the fifth intercostal space, or in rare cases behind the fifth rib or in the fourth space. In this line, the liver is overlapped by lung to the extent of about one inch. But in all cases of suspected hepatic disease, the upper margin of hepatic dulness ought to be determined in its entire course. In the *median line* in front, it usually corresponds to the base of the ensiform cartilage, or rises slightly above this. To the left of the median line it is difficult or impossible to define the upper limit of hepatic dulness from the lower boundary of the heart, the two being in apposition, but a line drawn from the upper margin of hepatic dulness in the median line to the apex of the heart will usually correspond to the line of separation. In the *right axillary line* (fig. 4), or a line falling perpendicularly from the centre of the axilla, the upper margin of hepatic dulness corresponds to the seventh intercostal space, or more rarely, to the seventh rib. In the *right dorsal line*, or a line falling perpendicularly from the lower angle of the scapula (when the arm is dependent), it corresponds to the ninth intercostal space, or the ninth rib (fig. 5).

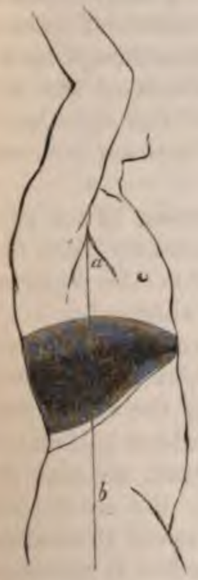

Fig. 4. Area of Hepatic Dulness, viewed from right side.

a-b, Right axillary line.

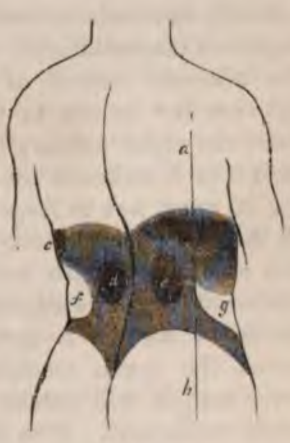

Fig. 5. Area of Hepatic Dulness, viewed posteriorly.

a-b, Right dorsal line. *c*, Splenic dulness. *d*, Left kidney. *e*, Right kidney. *f*, Descending colon. *g*, Ascending colon.

The lower margin of hepatic dulness may be determined by

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liver it is i
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on, and also, if diseased, by means of palpation. When
the lower margin of the liver cannot be distinctly felt
n the epigastrium. Even when the organ is diseased
a rule, less easily defined than the upper margin, being
scured by a distended condition of the stomach or intes
by fluid in the peritoneum. Hence it is always most
orily examined when the stomach is empty, and after th
have been freely moved. The liver may then be dis
ed from the intestines by the greater resistance it offer
ure by the hand. In the right mammary line, the lower
in health, usually corresponds with the margin of th
rch, or is half an inch above or below this; in the righ
line, it corresponds to the tenth intercostal space; an
right dorsal line, to the twelfth rib, although here it i
difficult to define it from the dulness of the kidney
epigastrium, the lower margin of the right and lef
ually descends to nearly half-way between the ensiform
e and the umbilicus.

ordinary extent of hepatic dulness, in an adult o
size, is 4 inches in the right mammary line, 4½ or
n the right axillary line, 4 inches in the right dorsal
1 3 or 4 inches in the median line anteriorly.

it must not be forgotten that, even in the same indi-
he liver is constantly liable to slight alterations in i
consistently with health. During the act of inspira
whole organ is slightly lowered—about half an inch—
upper surface is somewhat flattened, whereas durin
on the organ ascends. Again, in the erect positio
er margin will extend somewhat lower than when th
is recumbent. If in the mammary line it correspond
er margin of the costal arch in the latter position,
a half or a quarter of an inch lower in the former
ariations however are slight, and are not likely to em-
the diagnosis.

difficulties in diagnosis may sometimes arise from the
ies of the liver, as above defined, being greatly ex-
without any real enlargement of the organ. After
is often found that a liver which during life had been
to be greatly enlarged is even smaller than it ought
Hence, in all cases of suspected enlargement of the
is important to keep in view the possibility of its being
rious character.

CIRCUMSTANC

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III. *Rickets*, causing lateral distortion of the spine, and the deformity known as the 'pigeon breast,' may lead to apparent enlargement of the liver, owing to the organ being depressed and elongated in its vertical diameter from lateral compression. The resemblance to hepatic enlargement may be further increased by there being a disproportionate recession of the ribs immediately above the liver, as the result of which there is an apparent bulging of the hepatic region. Hence, in lateral distortion of the spine and in the 'pigeon breast,' care must be taken not to arrive at any hasty conclusion as to enlargement of the liver.

IV. *The practice of tight-lacing* may cause displacements and malformations of the liver, which may simulate enlargement, and give rise to difficulties in diagnosis. Tight-lacing may act on the liver in three ways, according to the situation, the tightness, and the duration of the constricting cause.

a. The liver may be displaced upwards or downwards, according as the pressure is applied below or above the organ. The precise situation where the pressure is applied will vary with the prevailing fashion of dress; but most commonly in this country the displacement is downwards, and this may be to such an extent that the lower margin of the liver reaches the ilium, and the organ appears to fill up the whole of the right side and front of the abdomen (fig. 6).

b. In consequence of lateral compression, the liver may be elongated in its vertical diameter, so that a larger portion of it is brought into apposition with the abdominal and thoracic walls. This is a very common result of tight-lacing (fig. 6). The narrower the lower portion of the chest, the greater will be the extent of liver opposed to the thoracic and abdominal walls.

c. When the pressure is exerted by a tight cord, it may produce deep fissures in the substance of the liver, as the result of which portions of the organ may be more or less detached, and may be felt as movable tumours separated from the hepatic dulness by tympanitic portions of bowel.

Apparent enlargements of the liver from tight-lacing are far more common than is generally believed. You cannot pay many visits to the post-mortem room without observing examples of this malformation, which accounts for not a few movable tumours in the abdomen that are a source of anxiety both to the patient and the medical attendant. Moreover, these acquired malformations of the liver, although most common in females, are occasionally observed in the male sex. I show you here the liver of a man with a deep furrow, from indentation of the ribs, which resulted apparently from the practice of wearing a very tight belt. I may also call your attention to the case of a man, aged 23, lately under your observation in the hospital, with a firm movable tumour in the epigastrium, which there was reason to believe was a portion of the liver partially detached from a similar cause.

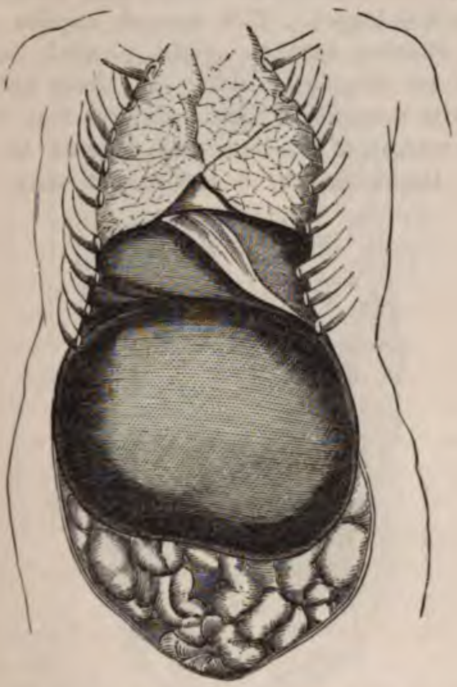

Fig. 6. Apparent enlargement of the Liver resulting from Tight-lacing. Modified from Frerichs. The Liver is depressed, and its vertical diameter elongated. A deep transverse furrow corresponds to the site of constriction.

Apparent enlargements of the liver from tight-lacing may usually be recognised by the following characters:—

1. Evident signs of tight-lacing in the walls of the chest and abdomen.

2. Occasionally the existence of a distinct transverse furrow in the substance of the liver, appreciable through the abdominal parietes on palpation.

3. The absence of symptoms of disease of the liver, or of serious disease in the chest or abdomen.

4. In the case of movable tumours from tight-lacing, their situation and the absence of any evidence of hydatid tumour or of disease of the gall-bladder will assist the diagnosis.

V. *Certain diseases in the chest* may cause great depression of the liver into the abdominal cavity, and lead to the idea that the organ is enlarged. This remark applies particularly to extensive effusion into the right pleural cavity, or to pneumothorax on the right side. In these affections the natural convexity upwards of the diagphragm may be reversed, and the lower margin of the liver may descend to the umbilicus (fig. 7). Depression to a less extent may result from intra-thoracic tumours, effusion into the left pleura or into the pericardium (fig. 8), or a dilated heart; and even in pulmonary emphysema and acute pneumonia[1] the liver may

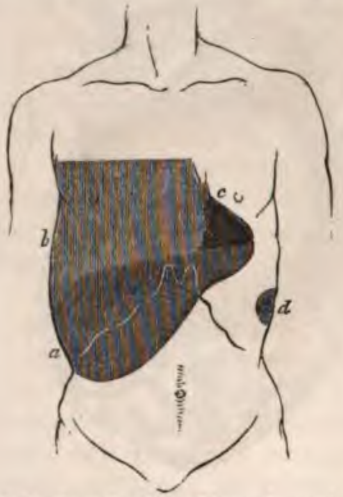

Fig. 7. Effusion into the Right Pleura depressing the Liver.

a, Hepatic dulness. *b*, Dulness from pleuritic effusion causing bulging of the right side of chest, and displacing the heart to the left; its upper margin horizontal. *c*, Cardiac dulness. *d*, Splenic dulness.

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[1] See a case of acute pneumonia of the right lung, referred to by Dr. Stokes in his work on 'Diseases of the Heart and Aorta,' p. 453. 'So great was the enlargement of the lung that the diaphragm and liver were pushed far down into the abdominal cavity.' Dr. Bright speaks of displacement of the liver downwards by pneumonic consolidation as a frequent occurrence (Abdom. Tumours, Syd. Soc. ed., p. 255); but Stokes regards it as exceptional, and this also is the result of my own observation. In extensive pneumonia, however, the liver is usually more or less congested, and enlarged accordingly.

be lowered to the extent of an inch or more. A dilated heart causes great depression of the liver far oftener than is commonly believed; not unfrequently from this cause the rounded upper surface of the liver becomes visible through the abdominal parietes below the ribs. In all cases, however, where the liver is depressed in consequence of disease in the chest, the descent of its lower margin is probably due to a combination of causes; for when there is disease in the chest sufficient to depress the diaphragm, there is usually also congestion with slight enlargement of the liver.

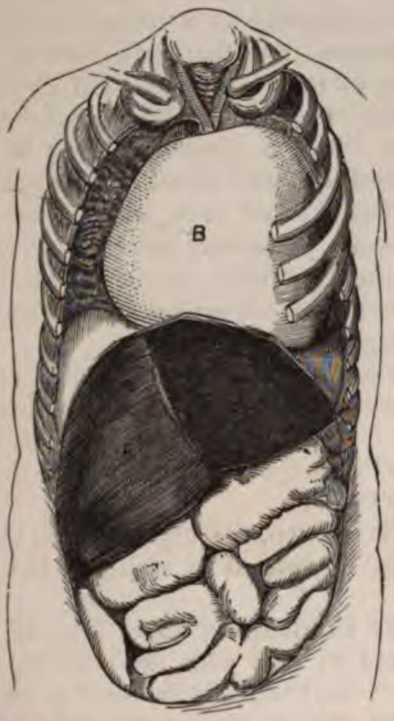

Fig. 8. Displacement of the Liver downwards by extensive Effusion into the Pericardium: after Sibson.

A, Liver. B, Pericardium greatly distended with fluid.

Apparent enlargements of the liver from the causes now referred to have the following distinguishing features:—

1. A previous history of pleurisy, pericarditis, bronchitis and emphysema, pneumonia, chronic cardiac disease, or of phthisis ending in pneumothorax. At the same time it is well

to remember that extensive effusion sometimes takes place into the pleura in a very latent manner.

2. A degree of dyspnœa far greater than would be accounted for by the amount of enlargement of the liver, even if real.

3. The physical signs of the various thoracic diseases above referred to. In the case of emphysema and pneumothorax, there is no difficulty in defining the upper margin of the liver, and in ascertaining that the extent of hepatic dulness is not increased, so that percussion will at once reveal the nature of the case. The signs of dilated heart also are usually sufficiently clear. But in pleurisy it may be impossible to say where the dulness of the pleuritic effusion ends and the hepatic dulness begins; and here, as in some forms of true hepatic enlargement, there may be bulging of the ribs and obliteration of the intercostal spaces (Cases I. II.). Under such circumstances there are several characters of considerable importance in diagnosis—viz.:

a. The bulging of the side of the chest is more uniform in pleurisy, and not abruptly limited to the lower part, as in diseases of the liver. An empyema, however, may be so circumscribed that the bulging is restricted to the lower part of the chest. (See Case I.)

b. In pleuritic effusion, the upper margin of the dull space is horizontal (fig. 7), instead of arched as in enlargements of the liver.

c. In pleuritic effusion, the upper level of the dull space will often be found to vary with the position of the patient. In enlargement of the liver, it is the same in all positions.

d. In pleuritic effusion, the lower margin of the liver does not ascend and descend with expiration and inspiration, which is the case in enlargements of the liver, unless there be firm adhesions to the abdominal wall.

e. Eversion of the lower costal cartilages would indicate hepatic enlargement, rather than pleuritic effusion. (But see Case II.).

f. When there is sufficient effusion into the pleura to cause downward bulging of the diaphragm, a depression may be sometimes observed between the lower margin of the ribs and the upper surface of the liver, which is not met with in hypertrophy of the liver.

Effusion into the pericardium will be recognised by the parent hepatic enlargement, the dimensions of the liver in every other direction being normal. Moreover, in both tumours of the kidney and of the omentum, when the patient lies on his back, the finger can usually be inserted between the ribs and the upper part of the tumour; there is often a clear space on percussion between the tumour and the liver; and the lower margin of the tumour does not ascend and descend with expiration and inspiration; while the diagnosis will often be assisted by the direction in which the tumour has grown, and by the history of the case. There are, however, certain difficulties in the diagnosis which must be kept in view. (Case VIII.) An omental tumour adherent to the liver may descend with it on inspiration. The kidneys, and particularly the right one, may be felt when healthy to descend slightly on deep inspiration, but this rarely occurs in the case of a renal tumour large enough to be mistaken for an enlarged liver. On the other hand, an enlarged liver may be prevented by peritoneal adhesions from moving with expiration and inspiration. Again, the liver may be pressed to the right by a distended colon or stomach, so as to simulate an enlarged kidney, or the transverse colon may pass in front of an enlarged liver, so as apparently to divide it into two distinct solid tumours.

Accumulations of fæces in the transverse colon also constitute a condition which it is often most difficult to distinguish from enlargement of the liver. Such cases are constantly occurring in practice, and it is well to bear in mind that, if you are to rely on the patient's statements, these accumulations are far from being necessarily associated with constipation. The resemblance to hepatic disease in these cases may be further increased by the hardened scybala imparting to the tumour a nodulated character like that of cancer, and by the development of such symptoms as jaundice, vomiting, and hiccup. The diagnosis of these cases from true enlargement of the liver must rest mainly on—

1. The occurrence of spasmodic pains like those resulting from obstructed bowels, &c.

2. The disappearance of the tumour, and the amelioration of the symptoms under such treatment as poultices and fomentations, purgatives, enemata, and belladonna.

Lastly:—

VIII. *Abnormal conditions of the abdominal parietes* may simulate enlargements of the liver.

Firm contraction of the bellies of the recti muscles, owing either to inflammation of the subjacent peritoneum or viscera, or, in cases of increased muscular irritability, to the mere application of the hand, is apt to be mistaken for hepatic enlargement, or tumour; and the difficulty is increased by the circumstance that the upper division of the rectus is sometimes larger on one (usually the right) side than on the other. It is distinguished by :—

1. The situation, size, and form of the apparent tumour corresponding to one of the divisions of the rectus muscle.

2. The sound on percussion being usually more clear and tympanitic than it would be over a solid tumour.

3. When the patient is made to sit up in bed, the swelling contracts and becomes thicker.

4. When the patient is placed on his back, with his shoulders raised and his thighs flexed on the abdomen, and his attention is engaged by conversation or by making him count, the tumour may disappear; and it will certainly do so if he be placed under the influence of chloroform.

The diagnosis may also be considerably embarrassed by an inflammatory swelling in the abdominal parietes over the liver. This has often been mistaken for an abscess of the liver itself. Several remarkable instances of this sort have come under my notice, in which, for some days, the diagnosis has been very doubtful. The following characters usually suffice to distinguish this condition from hepatic disease :—

1. The margin of inflammation and of dulness on percussion is ill-defined, and does not correspond to the boundary of an enlarged liver.

2. There is a greater amount of hardness and tightness of the superimposed integuments.

3. The constitutional symptoms are comparatively slight; rigors and profuse sweatings rarely occur, and there are no indications of severe hepatic derangement.

4. The lower margin of the swelling does not ascend and descend with respiration, but this character may hold good in adherent hepatic abscess.

Keeping in view these sources of fallacy, we proceed to consider the various causes of true enlargement of the liver.

The following cases illustrate some of the ways in which enlargement of the liver may be simulated. Cases I. and II.

are examples of a circumscribed empyema pointing below the ribs and causing great depression of the liver.

CASE I.—*Circumscribed Empyema of right side, displacing liver downwards, and simulating hepatic disease. Paracentesis below ribs and introduction of a drainage-tube. Recovery.*

Charlotte T——, aged 8, admitted into St. Thomas's Hospital May 22, 1872. Always delicate, but present illness had commenced six weeks before with rigors followed by pyrexia, loss of appetite, and emaciation; she had lain continually on right side. Her condition on June 1 was as follows: Liver appears enormously enlarged, upper part of abdomen distinctly bulging, especially on right side, and lower margin of liver descending to umbilicus. Abdominal veins unusually distinct and apparently much enlarged; no ascites and abdominal walls move freely in respiration. On right side of chest, dulness on percussion from liver to upper edge of nipple; above this clear percussion-sound and vesicular breathing, but below the nipple distinct circumscribed bulging of ribs and of intercostal spaces, with fluctuation between the ribs and also below them in right hypochondrium. Posteriorly, the dulness, absence of breathing, and bulging of intercostal spaces extend over lower two-thirds of right chest. Upper margin of dulness distinctly arched and has not ascended since patient's admission, but lateral bulging has increased much. Respirations 50; much pain and distress on slightest movement. Pulse 150; apex of heart felt between 5th and 6th ribs, half-an-inch outside left nipple. Temperature since admission has ranged from 99·6° to 102·8°; no rigors; for last three nights has perspired profusely. After an exploratory puncture, a large trocar was introduced into the swelling *below* the right ribs in front, and 53 ounces of pus drawn off; the first that came was thin, but the last thick and opaque. The opening was enlarged and a drainage-tube fastened in. The breathing was at once relieved, and it was observed during the operation that *the lower margin of liver ascended at least two inches, but that no change took place in level of upper margin of dulness in right chest.*

Next day child was much better. Pulse 114; respirations 30; temperature 97·8°.

June 22.—Continued to improve for a week after operation, but for last 13 days temperature has varied from 98·4° to 103°, and pulse from 120 to 150, and for several days pus discharged by tube has been fetid, although cavity has been washed out daily with Condy's fluid. Sleeps well; takes food well; and has had no rigors. Under chloroform a counter-opening was made at back between ninth and tenth ribs, and a perforated elastic drainage tube was passed through the two openings. About six ounces of very fetid pus came away during operation, and nearly a pint, also fetid, within next two days.

The cavity was now washed out daily with a solution of carbolic acid ($\frac{1}{100}$).

The quantity of discharge gradually diminished, until by the middle of August there was only a slight oozing of yellowish serum. The tube might have been now removed, but during my absence from town it was retained until Sept. 28; within a few days of its removal both openings healed. From a few days after counter-opening was made, patient steadily improved in general health, and when she was discharged on Nov. 1, she was plump and hearty, and for several weeks had been running about the ward. There was no difference on measurement, and scarcely any on inspection, between the two sides of the chest; if any, it was a slight excavation below right nipple. Hepatic dulness commenced at upper margin of sixth rib, 1½ inch below nipple, and extended 3 inches downwards, but not below edge of ribs. Clear percussion on right side posteriorly, with vesicular breathing down to normal level.

CASE II.—*Circumscribed Empyema, pointing at Epigastrium and depressing Liver. Paracentesis in Epigastrium. Recovery.*

On Dec. 23, 1875, I was requested to see a butcher, aged 40, who was supposed to have some serious disease of liver—hydatid or cancer. On careful enquiry, following history was elicited. For six or eight months he had complained of flatulent distension of stomach, but excepting this he had been in good health and attending to business, until beginning of November, when, after a chill, he was seized with severe pain across loins and general illness. After three days he sent for a doctor, who noted dulness over back of right lung and dry cough. Seven or eight days after this, during doctor's visit, he suddenly coughed up for first time a quantity of yellow matter; the expectoration continued for about a week, when it suddenly ceased. About Dec. 14 a swelling appeared at epigastrium with considerable pain, and about same time dulness at back of right lung receded.

At time of my visit, lower margin of liver was about one inch below umbilicus, its position not influenced by inspiration; in epigastrium was a circumscribed fluctuating bulging five inches in diameter, tender, but less so than it had been. Lower right costal cartilages everted. At base of right lung feeble breathing and some crackling sounds. Resp. 24. Pulse 100. Temp. 100°; no rigors or profuse perspirations. Appetite bad; sleep disturbed. An exploratory puncture was made in epigastrium, and a tablespoonful of thick fetid pus escaped.

Next day nearly two pints of pus were drawn off through a larger opening, a piece of elastic tube was tied in, and through this the cavity was washed out, first with three fluid ounces of a solution of chloride of zinc (30 grains to ounce), and subsequently with a weak would have resulted from a hydatid that had taken on inflammation. But peritonitis, extending over many weeks and leading to great accumulation of fluid in the peritoneum, is in most instances the result of cancer, and this was likewise indicated by the constant vomiting, the attacks of severe abdominal pain, the presence of a large tumour in the abdomen, and the emaciation; while the separate collection of fluid in the epigastrium might have been explained by a portion of the peritoneal fluid being encysted above the liver. The only symptoms pointing to aneurism were the syncope which preceded the attack of pleuropneumonia and the persistent dorsal pain.

CASE IV.—*Diffuse Aneurism of Thoracic and Abdominal Aorta, terminating in Chronic Peritonitis, with copious liquid Effusion.*

James D——, aged 42, coachman to a surgeon, admitted into Middlesex Hosp., Sept. 13, 1869, suffering from peritonitis. His father and mother had both been strong and healthy, and had both lived to over seventy: they had left a family of twelve children, of whom all were alive, and only one sister was delicate. Excepting the usual infantile diseases, patient had always enjoyed excellent health. He had formerly been engaged in the ice trade, and had then been accustomed to drink much beer and spirits, but for two years he had been a gentleman's coachman and had lived more temperately. On Jan. 1, 1869, while cleaning brougham, he suddenly felt very ill; he was able to lie down upon some straw, but he then became quite unconscious, and according to his master's (a surgeon) account he remained in a state of profound syncope for half an hour. Immediately after this he had an attack of right pleuropneumonia, by which he was laid up nine weeks; but in beginning of March he was able to resume work, and for nearly five months he drove out every day. Still all this time he complained of a severe and constant aching pain in back and right shoulder; his appetite was good, though not so good as before; he had no pain in abdomen, and no sickness.

At beginning of August, without any strain or unusual exertion, or in fact any obvious exciting cause, patient was suddenly taken with urgent vomiting and severe pain and distension of abdomen, with constipation; these symptoms lasted about a fortnight, when they gradually passed off and he recovered his appetite. On Sept. 4 sickness returned, and was attended by pain in stomach, but less severe than on former occasion. The patient, however, had severe pain in right shoulder and great thirst, and abdomen began to enlarge. After two or three days he felt better again, and for two days he was able to go out for a little, but on 10th he became worse, and since then he had suffered acute pain, and had vomited everything he swallowed. For nine days his bowels had been relaxed, and shortly before admission he had passed a considerable quantity of semi-coagulated blood from bowel, which his wife compared to clots passed after childbirth. He had never suffered from piles.

The patient's 'state on admission' was noted as follows: 'Very emaciated. Still suffers much from constant aching pain in back, but at present chief complaints are of pain and swelling of abdomen, and of inability to retain anything on stomach. Abdomen is considerably distended, tense, and tender; it measures at umbilicus 32 inches, this enlargement being due partly to fluid in peritoneum, but mainly to a tumour occupying centre and upper part of abdomen, and apparently connected with liver. Hepatic dulness in right mammary line $6\frac{1}{2}$ inches; in sternal line, it extends to 3 inches below umbilicus, and measures 10 inches. The lower 4 inches of this mass feel smooth and firm; its edge is well defined and does not ascend and descend with respiration; but above this, in epigastrium, there is distinct fluctuation with a circumscribed bulging over a space 5 or 6 inches in diameter. The fluid in this situation is evidently encysted and distinct from that in peritoneum; the thrill produced by tapping other parts of the abdomen is not propagated to it, and the bulging at epigastrium does not vary with position of patient. The abdominal walls scarcely move in respiration. Patient lies for the most part on right side, and says pain is always increased when he turns on left; he is also liable to paroxysms of severe abdominal pain irrespectively of position. No enlargement of abdominal veins; no obvious enlargement of spleen; no jaundice; tongue moist and white; says he vomits almost immediately after eating; bowels open three times to-day. Pulse 108, regular and feeble; apex of heart elevated, beating in nipple line; no abnormal pulsation or bellows-murmur anywhere over chest or abdomen. Occasional cough; respirations 36; perceptible respiratory movement almost entirely confined to left side of chest; over whole of right lung there is marked dulness on percussion, with very feeble tubular breathing; in front vocal resonance, and still more vocal thrill, are exaggerated; posteriorly they are absent. Skin covered with a clammy sweat; temperature 97·8°; slight œdema of feet and ankles. Urine contains $\frac{1}{20}$ (in volume) of albumen and much lithates.'

Patient was ordered ice, lime-water and milk, with brandy, a grain of opium twice a day, and poultices to abdomen. Subcutaneous injections of morphia were afterwards substituted for the opium pills.

Under this treatment diarrhœa was at once checked, and by Sept. 20 vomiting had also ceased, and patient's general appearance at first improved. The abdomen, however, slowly but steadily increased in size, and on Sept. 29 parietes were tense and glistening, and girth at umbilicus $33\frac{5}{8}$ inches. On Oct. 2 skin and conjunctivæ were slightly yellow, and there was bile-pigment in urine. On Oct. 18 girth at umbilicus had increased to 35 inches, and patient complained much of paroxysmal pain and tightness in abdomen, and of increasing weakness. Pulse was usually about 96, and temperature about 97·5°. On Oct. 22 there was a great increase of abdominal pain, attended towards evening by vomiting. He gradually sank, and died on morning of 23rd.

On *post-mortem* examination several quarts of turbid alkaline serum, having a specific gravity of 1020, and containing flakes of lymph and pus-corpuscles, in peritoneal cavity. Intestines and other abdominal viscera, and peritoneal lining of abdominal wall, coated with a thin layer of recent lymph easily peeled off. Nowhere any sign of tubercle or cancer. Liver extended downwards beyond umbilicus; its tissue was firm, but did not seem abnormal. Between liver and diaphragm was an enormous cyst, quite distinct from peritoneum, and containing fluid red blood. On opening chest, right lung was found to be everywhere firmly adherent, collapsed, dense, and carnified. Posteriorly, beneath thickened pleura, and extending as high as third rib, and outwards to angles of ribs, was another collection of fluid blood; and on further examination this blood, and that above liver, were found to be contained in a common sac, formed by a large aneurism of lower part of thoracic aorta originating immediately above diaphragm, and terminating below at origin of superior mesenteric artery. This aneurism consisted of a large rounded sac formed by a dilatation of entire aorta over two or three inches of its course. The arterial trunk entered this sac abruptly above, and passed off from it as abruptly below. The cœliac axis was given off from near lower end of sac. On right side the sac had given way, and blood was infiltrated between its coats for a short distance, but entire coats had also ruptured behind peritoneum, and blood escaping had dissected its way in different directions. The main portion was that seen at epigastrium above the liver, but it had also burrowed upwards behind right pleura. It contained several pints of blood, and its walls were formed partly by expanded coats of the vessel, lined with laminated fibrin at some places nearly an inch thick, and partly by diaphragm, liver, vertebræ, ribs, and pleura. The bodies of lower dorsal vertebræ were eroded and rough, and right ribs, at their origin, were also bared. The entire liver was displaced forwards, so that its upper surface was opposed to anterior abdominal wall; in this way organ appeared to be enlarged, but its weight was only 54 ounces. Heart not enlarged, and valves healthy; extensive atheroma of aorta. Left lung voluminous and healthy. Right kidney compressed and altered in shape by aneurism, and its cortex at point of contact opaque and white. Mucous membrane of stomach was intensely injected, and studded with hæmorrhagic erosions.

In Case V. it had been supposed that the patient was suffering from a tumour of the liver, but more probably this was simulated by a collection of fluid between the liver and diaphragm. If the tumour originated in the liver, it could only have been an abscess or a hydatid. The former was excluded by the absence of constitutional symptoms and the transparency of the tumour, to say nothing of the rarity of a large solitary abscess in a boy who had never left this country; while hydatid was rendered improbable by the rapid growth, the absence of any trace of echinococci in the contents, and the fact that a cup-shaped indurated base could be felt after the sac was emptied. The anatomical relations negatived a renal cyst, and a chronic abscess of the abdominal parietes was excluded by the absence of pyrexia, by the effect upon the tumour of coughing, inspiration, pressure, and position, by the eversion of the ribs and the displacement of the heart, and by the direction which the probe took after the bursting of the sac. The diagnosis which seemed most in accordance with all the facts of the case was that there was a circumscribed inflammatory effusion between the liver and the diaphragm; and it seemed possible that the 'ascites' which followed the varicella might have been tubercular, and that the injury to the back rekindled a fresh but localised inflammatory process. In reference to this case, the following remarks of Wilks and Moxon are of interest:—'We have seen several cases of large abscesses between the liver and diaphragm, or between the liver and stomach; the liver-tissue being only compressed by, and not involved in, the abscesses, which lay quite outside of it. Some of these were traced to injuries, but for others no cause could be assigned.'[1]

CASE V.—*Circumscribed Peritoneal Effusion between Liver and Diaphragm, depressing Liver.*

John J——, aged 10, was admitted into Middlesex Hosp. under my care, June 29, 1869. His father and mother were in good health; a brother and a sister had died of scarlet fever, and he had two brothers and two sisters alive and well. In infancy he had passed through measles and scarlet fever, and early in 1867 he had what was supposed to be an attack of varicella followed by temporary ascites. Since this last attack he had been rather weakly. About May 1868, he was struck on the back by a truck; he did not seem to experience any uneasiness from this at the time, but in September he became weaker, and began to complain of pain in the region of the liver, increased by taking a deep breath, and Dr. Schulhof, who then saw him, found a slight bulging of the right lower ribs, and noticed that the boy always leant to the right side. At end of December he contracted a second mild attack of scarlatina, and about middle of February, when Dr. Schulhof saw him again, there was a fluctuating painless swelling about the size of a hen's egg below right ribs, which could be forced up under the ribs when the boy lay on his back. From this time the tumour gradually increased in size without causing any pain.

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[1] Lect. on Path. Anat., 2nd ed., p. 446.

On admission, there was found to be a globular tumour in right hypochondrium, commencing immediately below, and not overlapping, the right ribs, and extending to about 3 inches below level of umbilicus (see fig. 9). It measured six inches over its convexity vertically, $6\frac{1}{2}$ inches transversely, and the circumference at its base was 14 inches. The cartilages of lower right ribs were slightly everted, and girth here was $\frac{1}{4}$ inch more than on left side. The tumour was painless and distinctly fluctuating throughout, and there was no induration at its base. It exhibited a slightly bluish translucent appearance, and the light of the sun or of a candle was distinctly transmitted through it. When patient coughed, an impulse was conveyed to tumour, and when he lay on his back and plaster of Paris was applied over tumour, with the object of taking a cast, a portion of tumour seemed to disappear beneath ribs; the tumour was always largest when he sat up. There was clear vesicular breathing at base of right lung, which descended to normal level both anteriorly and

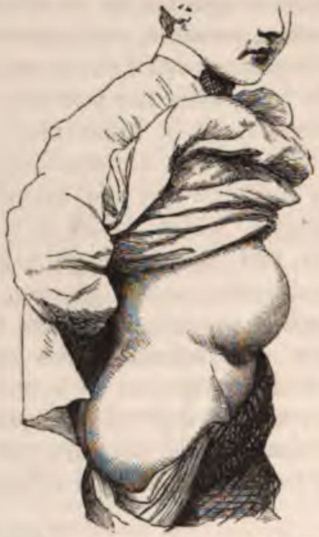

Fig. 9. Shows tumour in right hypochondrium of Case V.

posteriorly. The lower edge of liver could not be felt through tumour, which descended slightly on patient's taking a deep inspiration. There was tympanitic percussion noted between tumour and right kidney; no tenderness or curvature of spine; apex of heart beat between 4th and 5th ribs, immediately below left nipple. The boy's general health was good; he was rather thin and pale, but had no pyrexia, and ate and drank well; he had no sign of pulmonary, cardiac, or renal disease, and no jaundice.

On April 14 the tumour was punctured with a trocar, and 15 fluid ounces drawn off of thin pus, having a specific gravity of 1028 and separating on standing into two layers of about equal volume, the upper clear and straw-coloured, the lower opaque and yellow, and under microscope exhibiting pus-corpuscles and compound granular bodies, but no traces of echinococci or cholesterin. The chemical examination of the matter gave the following result:

Total solids	9·7	per cent.
Organic ,,	8·64	,,
Ash ,,	·86	,,
Chloride of sodium	·6	,,

The rest of the ash consisted of sulphate of soda and phosphate of lime.

The operation was followed by no constitutional disturbance, but in less than two days it was clear that the sac was again filling, and on April 29 the tumour was almost as large as before it had been emptied. On this day it was tapped a second time and 15 fluid ounces of fluid were drawn off, similar to that on first occasion, but with less sediment, and having a specific gravity of 1022. On May 7 a third tapping brought away 9 ounces of fluid more viscid than the former, and containing compound granular corpuscles adhering in flakes, but no distinct pus-corpuscles, and having a specific gravity of 1020. A fourth tapping brought away 7 ounces of fluid still more viscid, specific gravity 1019, and becoming perfectly solid on boiling. On each occasion after the tumour was emptied, a cup-shaped induration could be felt all round its base. After the fourth opening the tumour increased again very slowly, and on May 24, while patient was lying upon it, it opened spontaneously at a spot below where it had been tapped, but where for some time the integuments had been thin and dark. This spontaneous opening continued to discharge a clear viscid fluid containing white flakes until the patient left the hospital on June 29. A probe could be passed through the opening inwards, downwards, and outwards beneath the abdominal wall to the extent of an inch-and-a-half, but upwards beneath the ribs and above the liver to fully 3 inches. During the boy's residence in hospital he had gained flesh and improved greatly in strength and appearance.

On Oct. 5 he presented himself as an out-patient. His general health was still good. He brought with him a large quantity of calcareous flakes (not effervescing with nitric acid) which had come away from the opening shortly after he had left the hospital. The opening had not yet closed, but a probe could not be passed in any direction farther than 2 or 3 lines. Shortly after this the opening permanently closed, and one day in 1873 the boy presented himself at St. Thomas's Hospital in good health and having experienced no further trouble from the swelling.

CASE VI.—*Apparent Enlargement of Liver due to Peritoneal Adhesions.*

Elizabeth H——, aged 44, admitted into Middlesex Hosp., July 15, 1868, suffering from cardiac dropsy and other indications of disease of mitral valve. There was moderate ascites, and apparently great enlargement of liver, which could be felt as a solid tumour filling upper part of abdomen, and extending down to an inch below umbilicus, hard, smooth, and very slightly tender. Hepatic dulness seemed to extend upwards to about its normal level in front, but the presence of fluid in pleuræ made determination of this somewhat doubtful. A hard tûmour could also be felt obscurely below left ribs. The dropsy and dyspnœa gradually increased, and on Aug. 12 patient died.

At autopsy liver was found to be slightly, if at all, enlarged; but its upper surface was bound by firm adhesions to diaphragm and abdominal parietes to below umbilicus. Its capsule was much thickened and its structure was dense and fibrous; it weighed 61 oz. The spleen was also large, weighing 9 oz., and its capsule much thickened.

In the next case enlargement of the liver was simulated by a phantom tumour.

CASE VII.—*Phantom Tumour of Abdomen simulating Hydatid of Liver.*

On Feb. 17, 1869, Miss Hester D——, aged 11, a healthy-looking child, was brought for my advice as to tapping what was believed to be a hydatid of the liver. Two years before, on recovering from a low fever, a tumour had first been noticed in the epigastrium, which continued to increase for a year, and since then had been stationary. She had suffered from dyspeptic symptoms, but not from pain, and her general health had been good. There was a prominent rounded swelling extending from lower end of sternum to below umbilicus, rather straight on either side apparently from contraction of recti muscles. It was for the most part dull on percussion; surface smooth and elastic, but not fluctuating; no tenderness except at one spot over ensiform cartilage, where slightest pressure caused much pain. The degree of bulging varied somewhat, according as patient's attention was directed to it or not.

On Feb. 20 child was put under influence of chloroform; the tumour disappeared, and no tumour or enlargement of liver could be felt. When the effect of chloroform passed off, tumour returned; but under use of iron and belladonna it gradually diminished, and several years afterwards she was in excellent health.

Case VIII. illustrates the possibility of mistaking a large renal cyst[1] for a cystic tumour of the liver. The history of an injury was not incompatible with hydatid of the liver, for in many cases of hydatid the patients date their origin from an injury, which has been the means of drawing attention to a tumour already existing. There was no history of hæmaturia, of pus in the urine, or of other symptoms of urinary disturbance, such as can be elicited in the case of many renal cysts. Unfortunately the fluid drawn off during life was not examined for urea, but none was found in that which remained in the sac after death. Moreover, although Mr. Stanley has recorded two cases of renal cyst where the fluid contained urea,[2] none has been found in several other cases which are on record.[3] Lastly, although after death the ascending colon and coils of small intestine were found in front of the cyst, these could not be made out before paracentesis, when the cyst was tense. The operation was resorted to merely as a palliative, and contributed in no way to the fatal result; the inflammation of the sac and the secondary deposits in the lungs had commenced previously.

CASE VIII.—*Enormous Cystic Tumour communicating with Pelvis of Right Kidney, existing for eight years, and simulating Hydatid Tumour of Liver.*

Joseph O——, aged 16, was admitted into Middlesex Hosp. under my care Dec. 19, 1867. Eight years before he had been thrown with great force against a wall, injuring his back and right side. For a week after he vomited everything he swallowed, and altogether he was laid up for two months, but he never was observed to pass blood in his urine, or to have urinary symptoms of any sort.

He then went to school for a month, when he was seized with severe pain in his back and right side, for which leeches were applied.

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[1] Similar cases are recorded by Mr. Cæsar Hawkins (Med. Chir. Trans., vol. xviii. p. 175); Mr. Stanley (ib., vol. xxvii. p. 1); Sir Henry Thompson (Path. Trans., vol. xiii. p. 128); and Dr. H. Cooper Rose (Med. Chir. Trans., vol. li. p. 167).

[2] Med. Chir. Trans., 1844, vol. xxvii. p. 1.

[3] There was none in Dr. Cooper Rose's case, or in others referred to by Mr. Spencer Wells in the discussion upon Dr. Rose's case at the Medico-Chirurgical Society on May 12, 1868.

He was in bed for five months, and during this time he had freq
vomiting and nine fits of convulsions, the movements being limit
left side of the body. Shortly after this his mother noticed tha
right side had 'grown out,' and the swelling increasing she took
to the London Hospital, where he remained for four months,
where his general health underwent great improvement. His h
continued good, and he was able to go about, but the swelling sl
increased, until about a week before admission, when, after ge
thoroughly wet outside a cab, he was seized with severe pain in l
cough, and febrile symptoms.

On admission, patient was anæmic and emaciated, and compla
of cough and shortness of breath, and of great pain and tenderne
lower part of spine. Pulse 108; respirations 48 and thoracic; b
chitic râles over whole of both lungs, with dulness and friction
lower fourth of left. Tongue clean; appetite bad; temperature 10
No anasarca; and urine contained no albumen. But the most
markable feature about the boy was the enormous size of abdo
which measured 33½ inches at umbilicus, the bulging being gre
in right flank. This enlargement was almost painless, and was
dently due to an encysted collection of thin fluid on right side
tending from liver down into pelvis, and as far forwards as m
line, but clearly shut off from general cavity of peritoneum, as the
of abdomen was tympanitic in whatever position patient lay. He
dulness ascended to nipple in front, and to lower angle of sca
behind.

After admission, tumour increased in size, and dyspnœa bec
urgent that, on Dec. 23, it was resolved to tap cyst, which w
cordingly done by Mr. Hulke, midway between ribs and crest of
and 170 ounces of fluid drawn off. The fluid which first came
was clear, but of a brownish colour; its specific gravity was 10
it contained much chlorides, and about one-sixth of albumen
last two pints contained much pus, forming on standing a cre
posit, of about one-half of the entire bulk. No portion of t
contained either echinococci or hooklets.

At first the operation was followed by great relief to dysp
at no time afterwards had patient either rigors, profuse pers
pain in tumour, or albumen in urine. The prostration, ho
creased daily; tongue became dry; temperature varied fro
103·2°; much restlessness with sleeplessness and occasiona
and the signs of pleurisy at base of left lung noted befor
extended. He gradually sank, and died Jan. 2, 1868.

Autopsy.—No signs of recent peritonitis, but on right
domen, lying behind intestines, was a cyst, with thick f
about size of an adult human head. It was firmly attach
adhesions to under surface of liver, to false ribs, and
wall. It extended downwards to brim of pelvis, and s

middle line to left. Right kidney was expanded over its outer and posterior aspect, and the renal tissue was attenuated and wasted. The sac contained 65 ounces of thin pus; its inner wall presented a fibrous puckered aspect, with no trace of hydatid structure, and it communicated by three openings, oblique and valvular, but large enough to admit a full-sized catheter, with pelvis of kidney.[1] Right ureter was rather small, but pervious throughout; it ran for some distance in wall of cyst immediately beneath its lining membrane, and then passed down to bladder, which was quite normal. Upper part of right kidney was converted into a cicatrix-like fibrous tissue, intimately incorporated with cyst. Left kidney was double normal size, but otherwise normal. Liver fatty; spleen very large and soft. Recent pleurisy over lower lobe of left lung, which contained a patch of red hepatisation; and in lower lobe of right lung were several small patches of lobular pneumonia, with yellow centres. No pus in joints, and no sign of old fracture of ribs, or of disease of bodies of vertebræ.

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[1] It is remarkable that notwithstanding these openings the urine, up to the day of death, never contained any pus or a trace of albumen. A similar observation was made in the case recorded by Mr. Cæsar Hawkins and already referred to (p. 27). In that case also, although the cyst communicated with the pelvis of the right kidney, no urea could be found in the contained fluid, which is also said to have been devoid of albumen, although it contained pus.

LECTURE II.

ENLARGEMENTS OF THE LIVER.

TRUE ENLARGEMENTS OF THE LIVER: SUBDIVISION INTO PAINLESS AND PAINFUL: 1. THE WAXY, LARDACEOUS, OR AMYLOID LIVER; 2. THE FATTY LIVER; 3. SIMPLE HYPERTROPHY.

Bearing in mind the various circumstances under which I have told you that hypertrophy of the liver may be simulated during life, we are now prepared for considering those cases in which an increased area of hepatic dulness is due to real enlargement of the organ. And first of all it may be observed that enlargement is a character common to many different diseases of the liver, so that some classification will be a material aid in diagnosis. The late Dr. Bright, whose researches on diseases of the abdomen are scarcely less valuable than those on diseases of the kidneys, with which his name will for ever be associated, divided enlargements of the liver into two classes, according as their form was *smooth* or *irregular*.[1] But this subdivision is, in my opinion, open to the objection that in certain diseases (e.g. waxy liver) an enlargement which is usually regular and smooth may assume a lobular or nodulated character, whereas in others (e.g. cancer) an enlargement which is for the most part nodulated, may occasionally be perfectly smooth. A subdivision which appears to me to be, on the whole, preferable, is that into *painless* and *painful* enlargements. Painless enlargements are further characterised by an absence of jaundice and ascites, and by a chronic course; but in painful enlargements jaundice and ascites are common symptoms and the progress is more rapid.

Among painless enlargements we have the so-called amyloid liver, the fatty liver, hydatid tumour of the liver, and simple hypertrophy.

Among enlargements in which pain is a prominent symptom we have congestion, catarrh of the bile-ducts, obstruction of the common duct and retention of bile, interstitial hepatitis, pyæmic abscesses, tropical abscess, and cancer.

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[1] Abdominal Tumours. Syd. Soc. ed. p. 242.

There are other enlargements of the liver besides those now mentioned, such as tubercle, spindle-cell sarcoma, etc., the anatomical and clinical characters of which are less known. I purpose in a separate lecture to bring some of these rarer forms of enlargement under your notice, but in the first place we may consider in detail the distinguishing characters of the several forms of enlargement with which we are best acquainted.

I. THE WAXY, LARDACEOUS, OR AMYLOID LIVER.

The liver undergoes greater enlargement from the so-called waxy, or amyloid, deposit, than from any other disease, excepting, perhaps, cancer. I have known the liver of an adult affected with this disease weigh upwards of 180, instead of 50 or 60 ounces; and the liver, of which I show you here a portion, weighed one-seventh, instead of a twenty-fifth, of the entire body of the child from which it was taken. Enlargement of the liver due to waxy or amyloid deposit may be recognised during life by the following characters:—

1. The enlargement is often great, so that the liver fills up a large portion of the abdominal cavity.

2. It is uniform in every direction, so that the form of the organ is not essentially altered. The area of hepatic dulness on percussion is increased in the median, dorsal, and axillary lines, as well as in the right mammary. The increase is greater in front than behind, because in the former situation there is greater room for growth (figs. 10 and 11). It is increased in an upward as well as in a downward direction, although mainly in the latter, the lower margin often reaching the umbilicus, or even the right groin; but nowhere is there any outgrowth from the normal contour. The abdomen is enlarged, and often there is a visible bulging below the right costal arch and in the epigastrium, but rarely, if ever, is there any bulging of the ribs themselves; for waxy enlargement of the liver moulds itself over adjacent organs, and has little tendency to cause displacement of the ribs by excentric pressure.

3. On palpation, the portion of liver which extends below the margin of the ribs is very dense, firm, and resisting. There is no elasticity, and still less any feeling of fluctuation.

4. The outer surface is smooth, and the lower margin is somewhat more rounded than natural, regular, and free from indentation. In this respect, however, rare exceptions occur, an ignorance of which may lead to errors in diagnosis. Occasionally waxy deposit in the liver coexists with cirrhosis, or with what are known as syphilitic cicatrices, and then the surface of the organ may be nodulated, or even broken up into irregular lobes, separated by deep fissures, the existence of which may lead to the suspicion that the enlargement is due to cancer. In cases also of extreme enlargement there may be an exaggeration, so to speak, of the lobes into which the liver is naturally divided, deep fissures corresponding to the attachment of the ligaments. Some years ago I had an opportunity of observing a case of this sort under the care of Dr. Greenhow in the

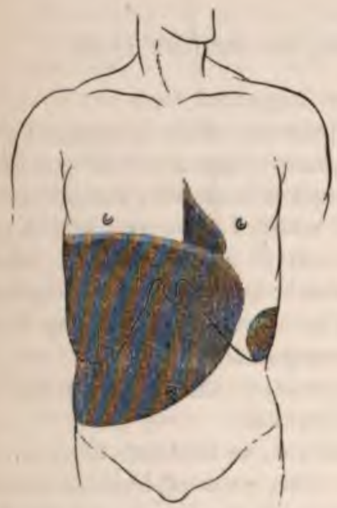

Fig. 10 shows the increased area of hepatic and of splenic dulness in the case of Henry D——: anterior view. Between the two is a space yielding the clear tympanitic sound of the stomach; and above the liver is the normal area of cardiac dulness. Compare this with fig. 3, which shows the normal boundaries of the liver and spleen.

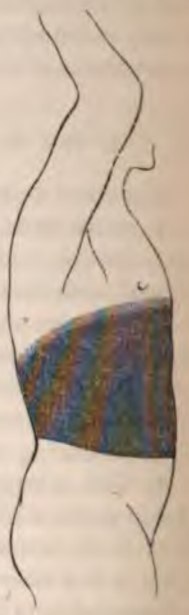

Fig. 11 shows the increased area of hepatic dulness in Henry D——: view on right side. The upper border is arched, and gradually falls towards the spine. Compare this with fig. 4.

Middlesex Hospital, the particulars of which I shall relate to you presently. Cases have also been recorded by Professor Frerichs, of Berlin, in which a waxy liver has presented a more or less lobulated form.

5. Waxy deposit in the liver has but little tendency to obstruct the portal circulation, and consequently ascites and enlargement of the subcutaneous veins of the abdominal wall are not common phenomena in its clinical history. When such indications of portal obstruction do occur, they are usually due to pressure exerted on the trunk of the portal vein by lymphatic glands in the fissure of the liver enlarged from waxy deposit. Occasionally, also, fluid is effused into the peritoneum as the result of general anæmia, concurrent disease of the kidneys, or secondary peritonitis.

6. Jaundice also is a rare symptom in waxy disease of the liver; and when it occurs, it is due, for the most part, to the pressure on the bile-ducts of enlarged lymphatic glands, or to the co-existence of catarrh of the bile-ducts.

7. Pain and tenderness are never prominent symptoms. The liver can be manipulated with impunity and the patient complains only of a feeling of weight or tightness in the right hypochondrium, or of uneasiness from the pressure to which the stomach and intestines are subjected. But occasionally, and particularly where there is a syphilitic history, there is an attack of acute pain from intercurrent peri-hepatitis. In the patient now under your notice, with paralysis of the right fifth nerve from syphilitic disease,[1] the liver and spleen, which are much enlarged from waxy deposit, were intensely tender for a time, owing to inflammation of their peritoneal covering; and in another case which I met with some years ago (Case X.), the enlargement commenced in India with severe pain in the right side, for which numerous leeches were applied, but the enlarged liver subsequently exhibited its usual painless character. Frerichs also has recorded a case where waxy liver supervened on protracted ague, and where 'the first symptom was persistent cutting pains in the side.'[2] Lastly, the presence of acute tenderness in waxy disease of the liver from the concurrence of peri-hepatitis was demonstrated in Case XI. by post-mortem examination.

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[1] Case in St. Thomas's Hospital, November, 1875.

[2] Diseases of the Liver, Syd. Soc. Transl. vol. ii. p. 200.

8. The growth of the tumour is slow and imperceptible. It often extends over several years.

9. Constitutionally, the symptoms are chiefly those of anæmia. There is no pyrexia; but the countenance is pale and sallow, the patient suffers from general debility, and the proportion of white corpuscles in the blood is somewhat increased.

Other characters of no small moment in diagnosis are derived from the spleen, the kidneys, the stomach, or the intestines being the seat of a similar morbid deposit to that producing the hepatic enlargement.

10. The spleen in most cases is enlarged, and often greatly, as well as the liver. The enlargement, like that of the liver, is uniform, hard, smooth, and painless.

11. As a rule, waxy disease produces enlargement of the liver before there is any evidence of its existence in the kidneys. Wetzlar found no albumen in the urine of any one of 18 patients suffering from syphilitic waxy enlargement of the liver.[1] When present, waxy disease of the kidneys has characters of its own, the presence of which in any case of hepatic enlargement would alone make it very probable that this enlargement was due to waxy deposit. These characters are :—

a. An increased quantity of urine. Not uncommonly the patient voids from three to five pints of urine in the twenty-four hours. This is the rule throughout the greater part of the course of the disease. Towards the termination only is the quantity diminished.[2]

b. The urine is of a pale lemon colour, of moderately low specific gravity (about 1014), free from any smokiness, and contains a considerable amount of albumen. In the early stage, however, there may be no albuminuria (Case XI.).

c. Casts of the renal tubes are often absent. When present, they may be of an epithelial or hyaline character, usually the latter, and most of them, from their size, appear to have come from tubes not denuded of their epithelium. These hyaline casts, so far as my observation goes, never yield the so-called amyloid reaction with iodine and sulphuric acid; but in exceptional cases this reaction may be observed in some of the cast-off renal cells.

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[1] 'Glasgow Med. Journal,' May, 1869.

[2] To Dr. Grainger Stewart, of Edinburgh, we are mainly indebted for pointing out the characters of the urine in waxy disease of the kidneys. My own observations coincide with his in every essential point.

d. During the greater part of the disease, when the urine is increased in quantity, there is no material diminution in the excretion of urea, and consequently the tendency to uræmia is much less than in other forms of kidney disease. Even in the advanced stage uræmic symptoms are comparatively rare, and death is more often the result of an exhausting diarrhœa.

e. According to Warburton Begbie, the urine contains uroxanthin in greater or less quantity, and when treated with an acid or exposed to the air, there is developed in it an indigo-blue, or indigo-red.[1]

The persistent secretion of a large quantity of urine containing much albumen by a person who has never had general anasarca will of itself warrant the presumption that he is suffering from waxy disease of the kidneys. In the contracted or gouty kidney there may also be no dropsy, and the quantity of urine may be increased; but here the specific gravity is remarkably low (often not exceeding 1010 or 1005) and albumen is usually present as a mere trace, or may be entirely absent.

12. The implication of the stomach and intestines in the waxy disease induces a tendency to vomiting and to obstinate diarrhœa from slight causes. Occasionally this diarrhœa is accompanied by tenesmus, and the patient may be thought to labour under dysentery; but post-mortem examination reveals no evidence of inflammation of the bowel.

13. The breath and skin in advanced cases often exhale a disagreeable odour, which is characteristic, and which Begbie has likened to that of musty indigo.

14. Here, as in many other maladies, the circumstances under which the disease usually makes its appearance are of considerable importance in diagnosis. There are certain conditions which pre-eminently favour the advent of waxy disease. Among them may be mentioned the following:—

a. Long-standing purulent discharge, such as is particularly apt to happen in connection with diseased bones or joints, dysentery, tubercular cavities in the lungs, and after surgical operations when the wound does not readily heal. In several cases of syphilitic ozæna I have met with waxy disease of the liver and other organs (Case XII.).

b. Constitutional syphilis. In a large number of cases of waxy disease the patients have been the subjects of constitutional syphilis, which appears to act as a predisposing cause independently of its inducing disease of the bones or protracted discharges, and independently of any abuse of mercury to which waxy liver was attributed by Graves and G. Budd.

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[1] Reynolds's Syst. of Med. iii. 966.

c. Tubercle of the lungs and of other organs must be regarded as a predisposing cause of waxy degeneration, although the enlargement of the liver common under such circumstances is oftener fatty than waxy. Of 52 cases of persons dying from tubercle, and whose autopsies I have recorded, the liver was fatty in 20, and waxy in 6, and in 3 of the 6 there was likewise caries of the bones. Still, of the 52 cases, 14 had waxy disease of either the kidneys, the liver, or the spleen, or 1 in $3\frac{5}{7}$. The proportion of tubercular males in whom waxy disease was found was more than double that of females. Thus, of 33 tubercular males, there was waxy disease in 11, or 1 in 3; whereas of 19 tubercular females, only 3, or 1 in $6\frac{1}{3}$ had waxy disease. It should be added that waxy disease from all causes is much more common among males than among females. Of 68 cases collected by Frerichs, 53 were males.

d. Many chronic diseases which impair the general nutrition seem to predispose to waxy degeneration, which has thus been met with as a sequel of protracted ague,[1] cancer, &c.

Treatment.—The following rules comprise those measures which experience has shown to be most useful in the treatment of waxy disease of the liver. In many cases, unfortunately, when the disease is already in an advanced stage, and when the kidneys and intestines are involved in the waxy degeneration, all treatment is of little avail, and the patient dies of exhaustion, which may be often ascribed to the copious drain of albumen in the urine or to the occurrence of profuse diarrhœa (as in Case XIII.). But, on the other hand, in not a few cases the progress of the disease appears to be arrested by appropriate treatment, and in some, as in Case X., there is reason for believing that the waxy deposit may be in great measure removed. In any case the danger is great in proportion to the extent to which the kidneys and intestines are involved.

I. *Prevention.*—The prevention of diseases in general has not yet received from the practical physician the attention which it deserves. The more we study the causes of disease, the more apparent it is that we possess a power in this direction which has hitherto been too much neglected. Bearing in mind the causes which we have found to lead to waxy enlargement of the liver, the means for its prevention will at once suggest themselves. First and foremost, it is always advisable to arrest as early as possible copious suppuration from any part of the body, and in particular from diseased bone, and, if necessary, to have recourse to surgical interference for this purpose. It may indeed be a question whether some of those operations, which what is called 'conservative surgery' has of late years substituted for amputation, from entailing protracted suppuration, have not sacrificed the life of the patient to the endeavour to save his limb. The death of the patient is ascribed to a bad constitution, which may, however, possibly be the result of internal disease engendered by the operation. In cases where the disease of the liver comes on in the course of phthisis, our treatment must be directed to the primary disease, and every means should be employed to arrest the purulent discharge from the lungs, the diarrhœa, and the exhausting sweats. Again, the symptoms of constitutional syphilis must always be met by appropriate treatment, and measures must be taken to prevent the condition of general cachexia which is apt to supervene on such exhausting diseases as ague and dysentery. Lastly, it may be mentioned that in cases where there is a copious suppurative drain from the system, alkalies have been proposed as a means of preventing the waxy deposit. Chemistry is said to have shown that the waxy material is dealkalised fibrin; and it is argued that as a large quantity of alkali passes off with the pus, the waxy deposit may be prevented by restoring this alkali to the system.[1]

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[1] See Lecture IV.

II. When waxy disease is already present, we must combat it by such measures as the following:—

1. The diet ought to be of as nutritious a character as is compatible with the digestive powers of the individual. A moderate allowance of alcoholic stimulants is generally useful. Considering the anæmic condition of the liver, alcohol is less likely to be injurious than in most other enlargements of the organ. When the disease is not too far advanced, and when the means of the patient permit, removal to a mild and equable climate is generally advisable.

2. *Alkalies.*—From my own experience I am not in a position to make any dogmatic statement as to the effects of alka-

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[1] Dr. Dickinson, Med.-Chir. Trans. Vol. L. p. 55.

neighbourhood of left hip, and sinuses continued to discharge until his death on Jan. 27, 1861. During life there was great tumidity of abdomen, obviously due to enlargement of liver, lower margin of which extended to below umbilicus, and surface of which was dense, smooth, and painless. Splenic dulness also increased, and the boy passed urine containing much albumen, but he had no dropsy. He was also liable to intercurrent attacks of diarrhœa, and tongue was preternaturally clean, red, and glazed.

Post-mortem examination.—Body extremely emaciated, joints being large in proportion to limbs. Total weight of body only 31 lb. 3 oz., or 499 oz. avoird.; length of body was $3\frac{1}{2}$ ft. Abdomen remarkably tumid and hard, particularly in right hypochondrium. Much swelling about left hip-joint, with numerous sinuses passing into bone. Left thigh flexed forwards and immovable. Entire head of left femur absent, and end of bone carious; acetabulum likewise diseased, bone being exposed and carious, and at one part deficient, so that there was an opening into pelvic cavity.

Head was remarkably large, its circumference being $21\frac{1}{2}$ in. Brain weighed $55\frac{1}{2}$ oz.; its structure normal. Each of lateral ventricles contained three drachms of serum, and at base were two fluid ounces. Membranes normal.

Heart and lungs normal.

Liver enormously enlarged and very dense. Its weight was 69 oz. avoird., or nearly one-seventh of weight of whole body, the normal ratio for a child nine years of age being only about 1 to 25. It reached as far as umbilicus, and moulded itself over the different organs in its vicinity. Its tissue was very firm, so that the organ retained its form when laid with its convex surface on table. Its external surface perfectly smooth and free from all adhesions, but exhibited impressions of adjacent organs. Its cut surface was of a grayish-pink colour and translucent, and presented a network of opaque yellowish streaks composed of fibrous tissue, apparently corresponding to outline of enlarged lobules, and enclosing the firm translucent material in its meshes. Iodine and sulphuric acid developed the so-called amyloid reaction in a marked degree. On microscopic examination, the hepatic cells appeared to be coherent into flat scales, and could not be isolated. The nuclei were distinct, but outlines of cell-walls were scarcely appreciable at many places, the nuclei appearing interspersed through a translucent homogeneous mass: at some places even the nuclei could not be distinguished. Towards circumference of lobules the cells were more distinct, and at some places contained an unusual amount of oil.

Spleen weighed $11\frac{3}{4}$ oz., and presented a dense, glistening surface on section, which became deeply tinged when treated with iodine and sulphuric acid.

Kidneys large, right weighing 5 oz., and left $5\frac{1}{4}$ oz. They were not at all dense, but, on the contrary, very flabby. Their capsules were non-adherent, and surfaces were perfectly smooth and pale yellow, with a network of injected veins. Cortical substance hypertrophied, pale yellow, opaque, and soft. Renal epithelium throughout kidneys loaded with fine molecules and oil-globules, and at many places uriniferous tubes appeared blocked up with oil. Iodine and sulphuric acid produced a decided tinging of minute arteries and Malpighian bodies in cortex.

Mesenteric and Peyerian glands slightly enlarged, and the application of iodine to mucous membrane of the bowel produced numerous brownish-red puncta, corresponding to the villi.

The co-existence of fatty kidneys with waxy disease of the livèr and spleen, in this case, is worthy of notice. It is to be observed, however, that even in the kidneys the minute vessels yielded the so-called amyloid reaction.

Many of you have had an opportunity of examining the patient whose case I am now about to relate.

CASE X.—*Constitutional Syphilis, followed by Symptoms of Waxy Disease of Liver, Spleen, and Kidneys.*

H. D——, aged 28, adm. Dec. 27, 1866, into Middlesex Hosp. As a young man he appears to have enjoyed good health, and to have been temperate. But six years ago he contracted syphilis, followed by buboes, which were opened, and scars are still visible in groins. Wound soon healed up, discharging only for about two weeks. He does not remember having had sore-throat or pains in bones. In 1858 he joined a cavalry regiment in India. With the exception of one or two slight attacks of diarrhœa, his health still kept good until about Nov. 1864, when he was seized with pain in right hypochondrium, which confined him to bed for six weeks. The pain was increased on taking a long inspiration; and he had leeches and blisters applied. At end of six weeks he returned to his duty; but his liver enlarging and his strength failing, he was discharged from the service, and arrived in England in June 1865. Since his return to England he has been able to earn his living as a labourer; but he has suffered each winter from cough, and expectoration occasionally slightly streaked with blood. Eight weeks before admission he lost his appetite and strength, and was sent as a case of 'fever' to London Fever Hospital, where he took mercury and iodide of potassium, with the object of reducing size of liver. On leaving the Fever Hospital he came here. He does not remember having had any form of fever in India, and at no time of his life has he had dropsy in any part of his body.

On admission, patient was thin and anæmic, and had a decided sallowness of countenance, without any jaundiced tint of conjunctivæ.

Over back were numerous small scars and copper-coloured discolourations. But what was most remarkable was the enlargement of liver, upper margin of which rose as high as fourth intercostal space, while lower margin reached as low as lower edge of umbilicus (see fig. 10, p. 32). The organ appeared large in every direction, its dimensions being as follows: In median line, 8¼ in.; in right mammary line, 9¾ in.; in right axillary, 6½ in.; in right dorsal, 5½ in. The upper margin of hepatic dulness was arched (fig. 11), that in axillary line being an inch lower than in right mammary; in right dorsal line it rose to eighth intercostal space, and from this it gradually fell towards spine. No bulging of ribs, and portion of liver below margin of costal arch very firm and resisting, not at all tender, and perfectly smooth. The only appreciable inequality was a transverse furrow situated 3½ in. above umbilicus, and apparently due to pressure of some article of clothing. Lower margin of liver considerably depressed when patient took a long breath, so that surface of organ was probably not adherent, or only slightly so. Dimensions of spleen likewise increased (see fig. 10); it did not project beyond the margin of costal arch, but dimensions of dulness were—vertically, 5½ in., and transversely, 6½ in., instead of 2 in. vertically and 4 in. transversely, as in normal state. No evidence of ascites or of anasarca. Appetite bad; tongue coated with a white fur, and for some weeks after admission a tendency to vomiting and diarrhœa, there being three or four relaxed motions daily. Patient did not complain of pain in abdomen, except of occasional transient attacks, which appeared to be due to flatulence. Chief complaint was of weakness in limbs.

Blood and urine were carefully examined. Blood was found to contain a slight but decided increase in proportion of white corpuscles, while many of the red corpuscles were of irregular outline and had a tendency to tail. Quantity of urine voided daily was ascertained for several weeks, and was always considerably above healthy standard; average quantity was from three to four pints, and occasionally there was more than four pints. Specific gravity varied from 1010 to 1015; urine always contained much albumen, but it was perfectly clear, of an amber colour, and without any palpable deposit. Microscopic examination for casts for the most part yielded negative results; on one occasion a few small hyaline casts were detected.

For first five days after patient's admission there was slight febrile disturbance. Pulse ranged from 110 to 120; temperature rose to 102°·4; moist and dry bronchial râles could be heard over back of both lungs. Patient had also sleepless nights, but without any rigors or perspirations. After this pulse and temperature were normal, and patient slept well; but a little coarse crepitus could generally be heard at bases of lungs. No evidence of heart disease.

The treatment up to March 13, 1867, consisted in mineral acids, bitter tonics, and a generous diet. At first, sulphuric acid and small doses of laudanum were prescribed, with the object of checking diarrhœa. On Jan. 9, nitric acid was substituted for the sulphuric, and was given with small doses of laudanum in the compound infusion of gentian. On Feb. 8 opium was omitted, and a grain of quinine substituted for the compound infusion of gentian. The diarrhœa, which had quite ceased, at once returned, but was again held in check by the restoration of laudanum to the mixture on Feb. 13. Under this treatment patient steadily and greatly improved. He had a good appetite, and was much stronger. His weight on admission was only 7 st. 10½ lb.; but on March 13 he had gained 16 lb.

April 3, 1867.—On March 13 the nitric acid was discontinued, and 15 minims of compound tincture of iodine substituted. After this patient continued to improve. He has now gained 20 lb. since admission. There has been no diarrhœa, and quantity of urine has diminished almost to natural standard. No material change, however, in size of liver.

April 29.—Patient was discharged from hospital to-day, greatly improved in strength and appearance. No diarrhœa, and urine was of normal quantity, with only $\frac{1}{20}$ albumen. Size of liver also greatly diminished, as will be obvious from following dimensions. In median line, 6 in.; in right mammary line, 7¼ in.; in right axillary, 6¼ in. Vertical splenic dulness only 4¼ in.

The circumstance of the enlargement of the liver, in this case, commencing in the tropics with acute pain, might have been thought to indicate abscess; but opposed to abscess were, the duration of the enlargement, its uniform character, its great density, the absence of fluctuation, and the fact of the patient having been able to work as a labourer for more than twelve months prior to his admission into the Fever Hospital. On the other hand, the physical characters of the hepatic swelling, the enlargement of the spleen, the excretion of a large quantity of very albuminous urine without any history of dropsy, the tendency to diarrhœa, the condition of the blood, and the syphilitic history, all pointed to waxy disease as the cause of the enlargement. As regards the pain, also, it may be stated that Frerichs records a case of waxy disease of the liver, in which 'the first symptom was persistent cutting pains in the side, and soon his strength diminished to such an extent that he felt it necessary to give up his work. Almost at the same time he observed a swelling in the right hypochondrium and epigastrium.' The cause of the pain was no doubt an intercurrent attack of peri-hepatitis, as was observed after death in Case XI.

CASE XI.—*Constitutional Syphilis, followed by Waxy Liver, Spleen, and Kidneys—No Albuminuria—Peri-hepatitis.*

Thomas S——, aged 23, a gardener, adm. into Middlesex Hosp. March 4, 1868. At beginning of 1866 he had contracted syphilis, and from Aug. 2 to Dec. 2, 1867, he had been a patient under my care, suffering from ulcerated throat, rupia, and periostitis of many of his bones, with attacks of pyrexia. Liver then was not enlarged. He came under my care the second time on account of urgent diarrhœa, which had lasted about a fortnight. He was then greatly emaciated, and had periosteal swellings on frontal bone, both clavicles, both tibiæ, bones of forearms, &c. Tongue dry and brownish; no appetite; much thirst; occasional vomiting; profuse watery diarrhœa; stools very offensive. Liver much enlarged, measuring 8 in. in right mammary line; enlargement uniform; surface smooth and hard, and for two days, but not before, intensely tender. No jaundice; no ascites; no obvious enlargement of spleen; no albuminuria; and no anasarca.

The diarrhœa was checked by a mixture containing tannic acid and a few drops of laudanum, but from time to time it returned. The tongue became dry, red, and fissured; abscesses formed over the jaw and in the hip; on the night of March 15 he had a severe attack of convulsions, from which he recovered, but on morning of the 31st he had a second fit, and remained unconscious until his death, eight hours after. Urine throughout contained no albumen; that passed on day before death was copious, and had sp. gr. of 1007.

Autopsy.—Liver greatly enlarged, extending down to umbilicus; its whole surface covered with a thin film of recent lymph, which could be easily scraped off; surface smooth, moulded over adjacent parts; structure extremely dense, and presenting the typical characters and reaction of waxy disease; weight, 131 oz. About one fluid ounce of amber-coloured gelatinous mucus in gall-bladder. Spleen, 12½ oz., firm and waxy, with appearance of sago-grains on section. Kidneys apparently healthy, but distinct 'amyloid' reaction of Malpighian bodies. Mucous membrane of intestine from jejunum to end of ileum presented marked 'amyloid' reaction of minute vessels in villi and elsewhere. Cranial bones greatly thickened, and diplöe filled with dense bony matter; a soft node over frontal bone, and corresponding bony surface rough and bare. Cerebral arteries had their coats thickened, but were not stained by iodine; about one ounce of fluid in lateral ventricles. Base of right lung carnified from compression by liver.

CASE XII.—*Disease of Nasal Bones—Ozæna and Epistaxis—Great Enlargement of Liver—Albuminuria.*

Philip A——, aged 41, labourer, adm. into Middlesex Hosp. Feb. 14, 1871. He stated that he had enjoyed good health until 16 months before, when he began to have an offensive discharge, with frequent bleeding from nose, and a troublesome cough. The quantity of blood lost was often considerable, and it would form large coagula in left nostril, which he would pull out. He became thin and weak, and had to give up work, and for nine months he had been an inmate of another hospital. He had never suffered from night-sweats, diarrhœa, or dropsy, but for two years he had been in habit of getting up two or three times in night to void water. He denied ever having had syphilis.

On admission, patient was thin, sallow, and anæmic, but there was no trace of jaundice or dropsy. He had a fetid discharge from nostrils, and left nostril was narrowed from projection into it of what appeared to be a portion of left nasal bone, quite bare and slightly loose. Over abdomen and legs were a good many small, copper-coloured, scaly spots. Abdomen large and prominent, girth at umbilicus being 31 in.; the prominence entirely due to an uniformly enlarged liver, the rounded lower margin of which could be felt two inches below umbilicus, and the hepatic dulness extending from nipple 10½ in. downwards. Surface of liver smooth, hard, and painless. Splenic dulness covered twice the normal area, but lower edge of spleen could not be felt projecting beyond ribs. Urine, while patient was in hospital, was examined almost daily; its quantity was increased sometimes to 97 oz.; its specific gravity varied from 1010 to 1015; and it usually contained albumen, sometimes as much as $\frac{1}{12}$ in volume, but no tube-casts. Slight flattening, dulness, and prolonged expiration below right clavicle. Pulse 80–100; heart displaced upwards, but in other respects normal. Tongue red and rather devoid of epithelium; appetite good; bowels regular. White corpuscles of blood considerably increased. During patient's stay in hospital, temperature in evening frequently rose two or more degrees, and once it was as high as 102·5°; he frequently complained of frontal headache and pains in limbs; for several days he had a slight attack of diarrhœa; and on April 11 he had a sudden and rather alarming attack of acute œdema of the glottis.

The treatment consisted in mineral acids and the pernitrate of iron, while nostrils were washed out daily with solutions of Condy's fluid, or sulphurous acid. When the frontal headache was most severe he took iodide of potassium, and the attacks of œdema of the glottis yielded speedily to leeches and hot poultices to throat, glycerine of tannin applied to rima glottidis, and pernitrate of iron internally. When patient left hospital, on May 24, he was considerably better. The liver was smaller, but hepatic dulness in right middle lobe still measured 9¼ in.; the albuminuria had for some time disappeared; proportion of white corpuscles in blood had diminished; evening temperature was normal, and discharge from nostrils was less.

CASE XIII.—*Syphilitic Necrosis of Lower Jaw—Albuminuria—Diarrhœa—Pleurisy and Pericarditis—Waxy Liver and Kidneys.*

John R——, aged 38, adm. under my care into Middlesex Hosp. Dec. 17, 1867. Six or seven years before he had contracted syphilis, and four years before he had been confined to bed for three months with a painful affection in joints, which he believed to have been rheumatism, and ever since he had been liable to pains in bones and joints. Twelve months before he had been a patient in same hospital with albuminuria and slight œdema of legs, and at that time the alveolar process of right side of lower jaw had exfoliated. Ten weeks before admission, he had been seized with cough, dyspnœa, and pain in right side of chest.

On admission, patient had an anæmic, chlorotic countenance, with slight general anasarca. The urine contained a very large quantity of albumen—about one-half—but no tube-casts; it was passed in considerable quantity, and had a specific gravity of 1015. Absolute dulness over whole of right lung, with all the signs of pleuritic effusion. Cardiac dulness also increased, but could not be isolated from that of right lung; sounds of heart feeble, but no abnormal murmur could be detected. Pulse 96. Tongue clean and red; breath extremely offensive; no appetite, and frequent vomiting. Hepatic dulness extended downwards uniformly, about two inches below normal boundary; above, it could not be well defined from dulness over right lung. The portion of liver projecting below right ribs was smooth and free from tenderness. Splenic dulness not increased. Patient suffered much from want of sleep.

Treatment proved of no avail in relieving patient's condition. On Jan. 2, profuse diarrhœa, with watery, very offensive motions, came on. This continued until patient's death occurred, on Jan. 7, by exhaustion rather than by coma.

On examining body, there was great thickening with firm adhesions of right pleura in front; posteriorly, right lung was separated from chest-wall by about thirty ounces of turbid fluid. Right lung extremely dense from fibroid change. Pericardium contained about twelve ounces of turbid serum, and surface of heart coated with a thick rough layer of rather firmly adherent lymph. Liver, spleen, and base of right lung all firmly adherent to diaphragm. Liver weighed 66 oz.; it was extremely dense, and presented the naked eye appearances and chemical reaction of waxy deposit. Spleen of natural size and rather soft. Kidneys of about normal size; their surfaces slightly granular; cortices extremely dense and pale, and the straight vessels and Malpighian bodies exhibited in a characteristic manner the so-called 'amyloid reaction.' Mucous membrane of small intestine intensely injected, but exhibited no 'amyloid reaction.'

In the following case which occurred some years ago in of the liver is painless from first to last. The organ can be freely manipulated with impunity, although in extreme cases the patient may complain of a feeling of weight or distension in the abdomen, increased by turning on the left side.

8. From the absence of symptoms, few opportunities are afforded of watching the growth of fatty enlargement of the liver, but this is usually slow and imperceptible.

9. The constitutional symptoms of fatty liver are few and not characteristic, and those which have been noted are often due for the most part to co-existing fatty degeneration of other organs, and more especially of the heart. General debility, great anæmia, and want of tone in the nervous and vascular systems are amongst the most prominent symptoms. The patient suffers from languor, is easily tired, and bears depletion or the inroads of acute disease badly. The late Dr. Addison described a condition of the integuments which he believed to be pathognomonic of fatty degeneration of the liver. 'To the eye,' he says, 'the skin presents a bloodless, almost semi-transparent, and waxy appearance. When this is associated with mere pallor it is not very unlike fine polished ivory, but when combined with a more sallow tinge, as is now and then the case, it more resembles a common wax model. To the touch, the general integuments, for the most part, feel smooth, loose, and often flabby; whilst in some well-marked cases all its natural asperities would appear to be obliterated, and it becomes so exquisitely smooth and soft as to convey a sensation resembling that experienced on handling a piece of the softest satin.'[1] These appearances are chiefly met with in females, and although they are far from being invariably present, yet in most cases of fatty liver the countenance and general integuments are more or less pasty and anæmic, and sometimes the skin appears greasy from increased action of the sebaceous follicles. Patients with fatty liver also suffer often from dyspeptic symptoms, such as flatulence, hypochondriasis, irregular action of the bowels—usually constipation, but occasionally profuse diarrhœa from slight causes.

10. Enlargement of the spleen is rarely present. The portal circulation is not obstructed to such an extent as to lead to enlargement of this organ from stasis of blood; and the spleen is not liable, as in waxy disease, to a deposit of the same material as that which causes the liver to enlarge.

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[1] Guy's *Hospital Reports*, First Series, vol. i. 1836, p. 479.

There are, however, certain other organs which are apt to undergo fatty degeneration as well as the liver, and the disease in each of these organs has symptoms of its own, which, when present, will throw light on the nature of the hepatic enlargement. Thus—

11. When there is concurrent fatty degeneration of the heart, in addition to the signs already enumerated, there are often—

a. A very feeble, or even inappreciable, cardiac impulse.

b. Very faint, or even inaudible, cardiac sounds, the first sound in particular being short and feeble.

c. A very slow, or a quick, feeble, and irregular radial pulse.

d. Attacks of vertigo, syncope, or pseudo-apoplexy.

e. Dyspnœa or sternal pain on slight exertion, and a feeling of sinking at the epigastrium.

12. When there is concurrent fatty degeneration of the kidneys, in addition to the signs already enumerated, there will usually be—

a. Urine below the normal standard in quantity, oftener turbid than clear, containing much albumen, and depositing numerous oil-casts.

b. A tendency to general anasarca.

c. Extreme pallor and pastiness of countenance.

13. As in waxy disease of the liver, the diagnosis will often be materially aided by attending to the circumstances under which the enlargement occurs. Many different conditions of the system may give rise to fatty enlargement of the liver, but most of them may be referred to one of the following heads:—

a. Large accumulations of fat beneath the skin throughout the body, in persons who for the most part are large feeders and lead indolent lives. It is in this condition that the heart is most likely to participate in the fatty change, and that you will expect to discover the symptoms of fatty heart already referred to. It is persons in this state who are most prone to die of rupture of the heart. In the 'Pathological Transactions' you will find several cases recorded in which patients died of rupture of the heart, and where not only was the heart found in a state of fatty degeneration, but the liver was enormously enlarged from fatty deposit, and there was a large accumulation of fat throughout the body.[1]

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[1] See particularly case by Dr. Quain, vol. iii. p. 262; and case by Mr. Pollock, vol. xv. p. 84.

b. Alcoholism.—Persons who drink immoderately of ardent spirits, particularly if they take little exercise, are very subject to fatty liver. Of thirteen persons who died of delirium tremens, Frerichs found the liver very fatty in six. Of two fatal cases of delirium tremens in which an autopsy was made by me in the Middlesex Hospital some years ago, there was considerable fatty enlargement of the liver in both: in one the organ weighed eighty-three ounces; in the other ninety-six ounces. It is under these circumstances that the kidneys often participate in the fatty degeneration; the quantity of fat also which some of these patients accumulate, notwithstanding the small amount of solid food which they consume, is remarkable. When the practice is persisted in, the fatty liver is apt to become complicated with cirrhosis.

c. Phthisis.—The great frequency of fatty enlargement of the liver in persons suffering from pulmonary consumption has been already referred to under the head of the waxy liver (p. 36). In consumptive females it is much more common than in males. In this disease, it is not a little remarkable that, while fat disappears rapidly from almost every tissue in the body, it should accumulate in such large quantities in the liver.

d. Other wasting diseases besides phthisis—such, for instance, as cancer,[1] simple ulcer of the stomach,[2] and chronic dysentery[3]—are likewise often attended by fatty enlargement of the liver.

It appears, then, that fatty liver is met with under two opposite conditions: one, in which there is an increased supply of material capable of being converted into oil, and where fat often accumulates in all the tissues of the body; the other, in which there is a rapid absorption of fat from all the tissues, with consequent emaciation. Its mode of production in the former case is sufficiently obvious; in the latter, the blood becomes loaded with oily matters derived from the patient's own tissues, and this oily matter is separated from the blood in its passage through the liver. The impaired absorption of oxygen in phthisis, interfering with the proper metamorphosis of the oil, accounts for fatty liver being more common in pulmonary than in other wasting diseases; and the greater frequency of fatty liver in women may be accounted for by their having in general a larger quantity, than men, of fat to be absorbed.

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[1] See case of cancer of the larynx, by Mr. C. Heath, Pathological Transactions, vol. xiii. p. 28; and case of extensive cancerous ulceration of groin, by Dr. Budd, Diseases of Liver, p. 299.

[2] Case by Mr. R. Robinson, Path. Trans. vol. iv. p. 133; and by Sir H. Thompson, id. vol. vi. p. 186.

[3] Case by Dr. Bright, in Hospital Reports, vol. i. p. 117.

Treatment.—It is not often that fatty enlargement of the liver causes such a derangement of functions as in itself to call for treatment. As a rule, treatment must be directed against the conditions in which the enlargement in question is known to occur.

1. When the disease is developed in persons who are large feeders and of indolent habits, the fat will usually disappear from the liver, as well as from the rest of the body, on the individual adopting an opposite mode of life. He must rise early and take active exercise in the open air, and live principally on lean meat, fish, bread, and green vegetables, with light claret, hock, or plain water to drink, and avoid butter, fat, oil, fermented drinks, strong wines, and all substances rich in starch or sugar. Under such a regimen, the fat will not only disappear, but the nutrition of the muscles will be improved, and the patient's strength increased. In cases, however, where there is reason to suspect the existence of fatty degeneration of the muscular tissue of the heart, the change of regimen here recommended must not be too sudden, and its effects must be carefully watched, while caution must be exercised in withdrawing the accustomed allowance of alcoholic stimulant.

2. When fatty liver is the result of alcoholism, a simple withdrawal of the cause will usually be sufficient to effect a diminution in the size of the liver.

3. Alkalies, alkaline carbonates, or compounds of the alkalies with the vegetable acids, in combination with some vegetable bitter, such as taraxacum or gentian, have generally been found useful for correcting the digestive derangements resulting from fatty liver; and if the bowels be constipated, recourse may also be had to occasional doses of the compound rhubarb, or colocynth pills of the Pharmacopœia, in combination with blue pill and extract of henbane, or to a dinner pill containing the watery extract of aloes and nux vomica. Eating large quantities of common salt with the food has sometimes appeared useful; and, when circumstances permit, it may be advisable to recommend a trial of the alkaline or saline mineral waters of Carlsbad, Marienbad, Kissingen, Ems, or Vichy.

4. The preparations of iron are often of great service in cases where there is marked anæmia, and those which are best suited are the ferrum redactum, the ferri et quiniæ citras, the ferri et ammoniæ citras, and the mistura ferri composita. They are often advantageously combined with alkalies. The chalybeate mineral waters of Tunbridge or Moffat, or of Spa, Pyrmont, or Schwalbach on the Continent, are useful for the same object.

5. Lastly, when the disease appears in the course of phthisis, it rarely calls for any special treatment, but its presence is a contra-indication to the use of cod-liver oil, or other oleaginous remedies.

In the following case, I had several opportunities of demonstrating to you in the wards the clinical characters of the fatty liver. The absence of albuminuria or of enlargement of the spleen made it improbable that the enlargement was due to waxy deposit.

CASE XV.—*Acute Phthisis—Fatty Liver.*.

Charles C——, aged 57, was adm. into Middlesex Hosp. under my care, June 11, 1867. He had enjoyed good health until about two months before, when he began to suffer from frequent cough, emaciation, and night-sweats, and subsequently from diarrhœa. On admission he was very thin and prostrate; frequent cough, with purulent expectoration; marked dulness for several inches below right clavicle, and coarse moist râles audible over whole of both lungs. Bowels very relaxed. Liver much enlarged; hepatic dulness in right mammary line measuring 7 in., and reaching fully 3 in. below margin of ribs. Enlargement was uniform; its outer surface smooth, but much softer and less resisting than that of waxy liver, and it was devoid of all pain or tenderness. No jaundice, albuminuria, or enlargement of spleen. The patient rapidly sank, and died on June 16.

On examination of body, both lungs infiltrated throughout with yellow tubercle, breaking down at apices into small cavities. At right apex pulmonary tissue had entirely disappeared. Numerous small ulcers, without tubercular deposit at edges or base in large intestine. Kidneys and spleen healthy. Liver much enlarged, weighed 78 ounces, smooth, pale yellow, opaque, and extremely friable; the secreting cells throughout loaded with oil.

III. SIMPLE HYPERTROPHY.

By 'simple hypertrophy' is understood an enlargement of the liver, due to an increased size of the lobules and an increased size or number of secreting cells, without any al tion of structure. The enlargement of the liver is uniform rarely great; and, as might be expected, it is not atte by any prominent symptom. The condition is comparat rare, and has still to be studied. It has chiefly been obse in:

a. Leukæmia; and in

b. Exceptional cases of saccharine diabetes.[1]

Hence, when the liver is found enlarged in either of t maladies without any obvious derangement of its funct simple hypertrophy may be suspected. It has been sugge that the enlargement of the liver arising from protra residence in hot climates may be of this nature; but in cases this is due to hyperæmia or to waxy disease (see Lec IV.).

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[1] See Frerichs' Diseases of Liver, Syd. Soc. Transl. vol. ii. p. 210. Accord Budd, the liver in diabetes is often unusually small, and the lobules shrunken the quantity of oil being below the normal standard (Diseases of Liver, 3rd ed. p. In many cases after death from diabetes the liver presents nothing abnormal.

LECTURE III.

ENLARGEMENTS OF THE LIVER.

IV. HYDATID TUMOUR.

THE fourth form of painless enlargement of the liver is that which is due to the presence of hydatid tumour. Although the disease is less common in this than in some other countries,[1] I have frequently had opportunities of pointing out to you its clinical characters, which are mainly the following :—

1. The enlargement may be very great, so as to fill the greater part of the abdominal cavity, or reach upwards to near the clavicle, but in its earlier stages the hydatid may form a globular tumour at one part of the liver, not larger than an orange; or from its situation and size it may altogether elude observation.

2. Unlike any of the enlargements already considered, it is not uniform in every direction, but usually it follows one direction in particular; so that the natural form of the liver is greatly altered (figs. 12 and 13). If it grow upwards, the natural arched outline of the upper boundary of hepatic dulness will be exaggerated; if it grow downwards, the lower boundary

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[1] Out of 2,100 post-mortem examinations recorded at the Middlesex Hospital between April 19, 1853, and August 25, 1863, hydatids were found in only 13, or once in 161 cases; and in only 7 of the 13 cases, or once in 300 cases, were they the cause of death. But in Iceland, Eschricht has calculated that about one-sixth of the entire population are afflicted with hydatids: and according to Hjaltelin, they are found in nearly one-fifth of all adult dead bodies. (*Brit. Med. Journ.* Aug. 14, 1869.) In Australian hospitals, hydatids are the cause of one in every 139 deaths. (Macgillivray, *Australian Med. Journ.* March 1867.) On the other hand, hydatids are much rarer in Scotland than in England. Dr. Scott Orr has searched the records of the Glasgow Royal Infirmary from the earliest periods, but has only found three cases, one in the mamma, and two in the liver. (*Glasgow Med. Journ.* Jan. 1876.) Dr. Gairdner also states that among many thousand dissections, which he had either performed or seen performed, during his connection with the Edinburgh Royal Infirmary, in only one instance had a hydatid been found in any part of the body, and that was in the upper part of the right lung. The patient came apparently from Newcastle. (*Clinical Medicine*, p. 431.) Can this immunity be due to the non-importation of foreign sheep into Scotland?

of hepatic dulness will be found to be natural at some places, while at others there is an abrupt protuberance or tumour (see fig. 12). Not unfrequently it takes a lateral direction, and causes more or less bulging of the ribs; and then the disease is apt to be mistaken for empyema, which is distinguished by the characters already enumerated (see page 12). It is the right lobe of the liver from which the tumour commonly grows.

3. It is neither dense nor doughy, but elastic, or even fluctuating. If the hydatid be deeply seated, with much hepatic tissue separating it from the outer surface, the tumour will be only elastic; but if it approach near to the surface there will be distinct fluctuation, with a thrill as from fluid, on palpation. Occasionally there is the sign known as '*hydatid vibration.*' This is a peculiar trembling sensation, experienced when three fingers of the left hand are laid flat on the tumour, and the back of the left middle finger is struck abruptly with the point of the middle finger of the right hand. This sign is not due, as is commonly stated, to the secondary cysts in the interior striking the wall of the parent; it may be detected in barren hydatids,[1] and it is not peculiar to hydatid tumours. It is elicited when any large cyst, with thin tense walls and watery contents, is treated in the manner above described. But, inasmuch as the only tumours of the liver answering to these characters are hydatids, the sign referred to, when present, is of considerable value in the diagnosis of hydatids in the liver. Unfortunately, in a large proportion—probably the majority—of cases of hydatid tumours of the liver, it is altogether wanting.

4. The surface of the tumour is smooth, and free from irregularities of every sort. In rare cases, when there are several distinct cysts projecting from the surface of the liver, this organ may appear through the abdominal parietes to have somewhat of a lobulated character, which may occasion considerable embarrassment in diagnosis. The possibility of this source of fallacy must be kept in view.

5. Ascites, œdema of the lower extremities, enlargement of the superficial veins of the abdomen, and hæmorrhoids are not distinguishing characters of hydatid enlargement of the liver. Their occurrence in rare cases must be regarded as in some measure accidental, and due to compression by the tumour of the trunk of the portal vein, or of the inferior vena cava, or of the

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[1] See also Trousseau's Clin. Lect. Syd. Soc. Ed. iv. 275.

iliac veins. Care must be taken not to mistake for ascites an enormous hydatid tumour projecting down from the liver and filling the fore-part of the abdominal cavity. This is distinguished by a history of growth from above downwards, and by the portions of the abdomen yielding tympanitic percussion not being the most elevated in any position of the patient. For instance, when the patient lies on his back, there may be dulness on percussion and unmistakable evidence of fluid in the most elevated part of the abdomen, while in both flanks the percussion is tympanitic (see Case XXXVIII.). When hydatid tumour of the liver co-exists with ascites, and no opportunity has been afforded of examining the patient prior to the ascites, the diagnosis will be extremely difficult, if not impossible.

6. Enlargement of the spleen is not a common consequence of hydatid enlargement of the liver, but may occur under conditions similar to those which occasion ascites. In very rare cases, the spleen may be enlarged from the presence of secondary hydatid tumours.

7. Jaundice is also an exceptional, and, so to speak, accidental symptom of hydatid enlargement of the liver. When present, it is due to pressure by the tumour on the common bile-duct, which is thereby narrowed or even obliterated, to catarrh of the bile-ducts, or to the bursting of the tumour into the ducts, which become obstructed by its contents. I show you here a specimen taken from the body of a gentleman under my care, in whom jaundice was due to the last of these causes (Case XXXIV.), and you have had opportunities of studying the symptoms in similar cases, which have proved fatal in the hospital (Cases XXXI to XXXIII.).

8. Enlargement of the liver from hydatid tumour rarely interferes with the functions of the kidneys, and hence we do not meet with those alterations in the urine so common in waxy, and of frequent occurrence in fatty, enlargements. In rare cases, however, the kidneys also may be the seat of hydatids, or pyelitis may be induced by the pressure of a large hydatid tumour of the liver on the ureter. Under these circumstances the urine may contain large quantities of pus, as happened in a patient who was under my care in this hospital a few years ago, and the particulars of whose case I shall narrate to you presently (Case XLII.). Occasionally the urine contains albumen, apparently from pressure on the renal vein, as it disappears after the cyst has been tapped.

9. The growth of a hydatid tumour is slow and imperceptible, and, when the tumour is large, it has usually existed for years before the patient has recourse to medical advice. Dr. Budd mentions the case of a lady who died at the age of 73, and in whose body two hydatid tumours of the liver were found, which there was reason to believe had existed since she was eight years old.[1]

10. The latent character of hydatid enlargement of the liver is one of its chief characteristics. It often attains a great size without causing any pain or uneasiness, and often indeed without the patient being aware of its existence,[2] and unless the sac be inflamed on its inner or outer surface, the tumour can usually be manipulated freely without causing tenderness. The first local indications of its presence are those resulting from pressure on adjoining parts, a feeling of weight or distension, of dragging pains, or of embarrassment of the breathing. Then, and not till then, it may become the seat of occasional attacks of acute pain and tenderness, in consequence of inflammation of the superimposed peritoneum. But now and then, a comparatively small tumour causes pain, by projecting in a direction where there is little space for its growth, or by compressing some nerve (Case XXI.).

11. There may, in like manner, be an absence of all constitutional symptoms. Even when of large size, the tumour often does not interfere with the functions of the liver. There is no pyrexia or impairment of the general health, and the chief symptoms are those due to pressure on adjoining organs, and interference with their functions. Some years ago a patient came to me complaining of cough and shortness of breath, and fearing that she was consumptive. On examining the chest, I found an enormous hydatid tumour of the liver compressing the right lung, and causing great bulging outwards of the ribs, as well as a prominent tumour in the abdomen. The patient had suffered nothing except the cough and dyspnœa, and was not aware of the existence of any tumour (Case XLII.). Instances also are not uncommon of patients who have died from acute inflammation excited by the bursting of a large hydatid tumour of the liver, who, previous to the

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[1] Diseases of Liver, 3rd ed. p. 433.

[2] Of 17 specimens of hydatid of the liver in the Berlin Pathological Institute, 13 had given rise to no symptoms. (Zeller, in Ziemssen's Cyclop. of Med. vol. iii. p. 593.)

attack of fatal inflammation, have been thought to be in perfect health (Case XXXIX.).

12. The diseases most readily confounded with hydatid of the liver are abscess, distended gall-bladder, effusion into the right pleura, aneurism, cancer, cystic tumour of the kidney, phantom tumour, and ovarian cyst.

a. The absence of symptoms, both constitutional and local, and the slow grówth of hydatid tumour form a marked distinction between it and abscess, which, so far as its physical characters are concerned, is the form of hepatic enlargement most closely resembling hydatid. There is one source of fallacy, however, which must be kept in view, although an accurate diagnosis under the circumstances would not materially modify the prognosis or the treatment. A hydatid tumour of the liver occasionally inflames and suppurates, and then it may present all the constitutional and local phenomena of abscess. The diagnosis of this condition must depend entirely on the patient's previous history—the fact of a painless tumour having long preceded the symptoms of abscess, the absence of exposure to the ordinary causes of tropical abscess, and the absence of any history of dysentery.

b. A distended gall-bladder may closely resemble a pendulous hydatid of the liver, and may also be free from pain. It is recognised by its shape and position, by its development being usually preceded by attacks of biliary colic, and by the fact that in most cases there is jaundice, from obstruction of the common duct. It must not be forgotten, however, that when a hydatid opens into a bile duct, the contents of the cyst in their passage along the duct may give rise to all the phenomena of biliary colic, including jaundice. Sir Thomas Watson has recorded a remarkable instance of this sort,[1] and several have come under my own notice, the particulars of which I shall presently relate to you.

c. Extensive effusion into the right pleura, with bulging of the ribs and obliteration of the intercostal spaces, may closely simulate a large hydatid tumour; but, on the whole, a hydatid of the liver is more likely to be regarded as an example of pleuritic effusion, than pleuritic effusion mistaken for hydatid. The hydatid is mainly distinguished by its insidious growth, and by the absence of constitutional symptoms. The chief physical distinction is derived from the upper boundary of the

[1] Lectures, 5th edition, 1871, II., 632; also Trousseau, op. cit. IV. pp. 237, 276.

dull space. In pleuritic effusion this is horizontal (page 10); in hydatid tumour it is arched, the convexity of the arch varying in its position with that of the tumour in different cases, but always fixed in the same patient. The possibility, however, of a hydatid of the liver co-existing with pleuritic effusion must not be lost sight of (see Cases XXXIX., XL.); under such circumstances the diagnosis may be extremely difficult. Moreover, an encysted pleurisy may simulate hydatid by producing a circumscribed bulging of the lower ribs, notwithstanding what Trousseau[1] says to the contrary (see p. 16).

d. An aneurism of the abdominal aorta, or of the hepatic artery, may present a smooth, globular tumour, very like that of a hydatid. Its main distinctive characters are pulsation, bellows-murmur, and the fact that it is usually the seat of acute neuralgic pains, owing to pressure on the branches of the solar, or of the hepatic, plexus. An aneurism of the hepatic artery is further distinguished by its being accompanied by jaundice from compression of the bile-ducts.

e. Cancer of the liver is mainly distinguished by its irregular surface, tenderness and hardness, and by the absence of elasticity or feeling of fluctuation. The diagnosis may be embarrassed by the circumstance that several hydatid tumours projecting from the surface of the liver may impart to it an uneven surface (Case XLV.), or that the nodules, or an extensive infiltration, of medullary cancer may exhibit a degree of elasticity approaching to fluctuation, or that in rare cases a large cyst may be developed in the liver in conjunction with cancer (Case XCVI.). Under such circumstances, the diagnosis of hydatid must mainly depend on its slower growth and on the absence of constitutional cachexia.

f. Renal Cyst. I have already had occasion to refer to the difficulties in distinguishing between a large renal cyst and an enlarged liver (page 14). A renal cyst is distinguished from a hydatid of the liver by:—1, its place of origin and direction of growth; 2, the presence of colon in front of the cyst; and 3, its position being little, if at all, influenced by deep inspiration. The characters of the fluid obtained by exploratory puncture will not assist you much in diagnosis. There will of course be no echinococci or fragments of hydatid cyst, but you may fail to find these in the fluid drawn off from a hydatid. On the other hand, the fluid may have a specific gravity of 1010, and may contain no urea, but abundance of chlorides with pus and albumen, characters which are quite compatible with the fluid from an inflamed hydatid (Case VIII.).

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[1] Op. cit. iv. 267.

g. A circumscribed Phantom Tumour in the epigastrium or right hypochondrium may be mistaken for a hydatid. Not long ago I saw a case in my private practice where this mistake was committed (Case VII.). It is distinguished by the absence of fluctuation or vibration, and by the circumstance that the tumour disappears when the patient is put fully under the influence of chloroform.

h. Ovarian Cyst. There is rarely any difficulty in distinguishing between a cyst of the liver and an ovarian cyst. The main distinguishing characters of a hepatic cyst are:—1. Its growth from above downwards; 2, the hand can be passed between its lower margin and the brim of the pelvis; 3, its lower margin is depressed by deep inspiration; 4, the enlargement is usually greater above the level of the umbilicus than below; 5, the examination of the fluid obtained by exploratory puncture would at once remove all difficulty in the diagnosis. There may, however, be some difficulty when a hepatic cyst is seen for the first time after it has attained a large size; and not long ago a case was recorded in one of the medical journals where the operation of ovariotomy was commenced in what proved to be a cyst of the liver.[1]

If there be any doubts as to the nature of the case, they may in most cases be removed by an exploratory puncture. The fluid which escapes from a hydatid, even if it contain no echinococci or shreds of striated hydatid membrane, will reveal its nature with absolute certainty. If the sac be not inflamed, it is limpid, when running in a stream, with a slight opalescence when viewed in bulk; it is alkaline, and has a specific gravity of 1009 (1007–1011); it contains neither albumen nor urea, but throws down a copious white precipitate with nitrate of silver, owing to its strong impregnation with common salt. These characters apply to no other fluid in the body, whether healthy or morbid.[2] Even if the case should turn out to be an aneurism or a cancer, no harm is likely to result from an exploratory puncture.

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[1] Brit. Med. Journ., Dec. 5, 1874. In this case, the slow progress, the absence of irregularities from the surface, and the decided fluctuation were, in my opinion, no arguments against hydatid tumour, as was contended.

[2] The contrast between the fluid in the hydatid cysts described in Case XLIV. and the surrounding peritoneal fluid, in which they were floating, is worthy of notice. According to Naunyn, hydatid fluid has a specific gravity of 1010 to 1013, and contains some albumen, but this is contrary to my experience except when the sac is inflamed, or blood has become mixed with it.

Modes of Termination of Hydatid Tumours of the Liver.—It may be thought that a tumour which causes so little inconvenience, that even when of large size the patient himself may be ignorant of its existence, requires little interference in the way of medical treatment. In reference to practice it is therefore important to have a correct knowledge of the natural modes of termination of hydatid tumours of the liver. The chief of these are as follows:—

Spontaneous Cure.—In the first place, there can be no doubt that some of these tumours undergo a spontaneous cure. The parasite may die from calcification of the parent cyst preventing further growth, from inflammatory action lighted up by the entrance of bile or by some other cause, or from the secondary vesicles increasing out of all proportion to the fluid in which they float (Case XLVII.); the parent cyst slowly shrivels up, and in place of the hydatid we find a putty-like material, the real nature of which is disclosed by its containing shreds of the striated hydatid membranes or hooklets of echinococci. But, unfortunately, this favourable result is confined for the most part to tumours of so small a size that they are not recognised during life. Case XLVIII. is a remarkable exception to the general rule in this matter. Watson also (*op. cit.* ii. 635) diagnosed a hydatid cyst in a young nobleman, who died 22 years afterwards, when a shrivelled hydatid was found in the liver. When the tumour is sufficiently large to give rise to symptoms and be diagnosed, such an event is so exceptional that it cannot be calculated on. The tumour then continues to increase in size. Its growth may be slow; it may extend over years; but almost as surely as the tumour grows will it one day burst, or lead to an equally dangerous though less sudden result. Even a cyst which has undergone apparently a spontaneous cure, may, as Dr. Church has shown, light up fatal inflammation.[1] The directions in which a hydatid tumour of the liver may burst are very various, and the danger will vary accordingly.

1. *Into the Pleural Cavity or Pulmonary Tissue.*—This direction is more common than any other. It is almost always the right lung and pleura that are invaded. When the contents of the hydatid are discharged through an opening in the dia-

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[1] Treatment of Hydatid Tumours of Liver, 1868. See also Case XLI.

phragm into the pleura, acute and almost invariably fatal pleurisy is the result.[1] After death the pleural cavity is found full of pus containing numerous hydatid cysts (Case XXXIX.). Trousseau has recorded cases in which an empyema thus induced has subsequently burst into a bronchial tube. Fatal pleurisy may also result from a hydatid tumour of the liver, without any perforation of the diaphragm.[2]

If adhesions form between the diaphragm and the base of the right lung prior to the bursting of the hydatid, the contents of the latter may escape along with bile by the bronchial tubes, and the patient may recover;[3] but even here, in most cases, fatal inflammation or gangrene is set up in the lung,[4] or the patient dies of suffocation from occlusion of the bronchi by hydatid cysts, or of exhaustion, owing to profuse discharge from one or several cavities excavated in the lung.[5] From Case XLI., also, it will be seen that an obsolete hydatid cyst of the liver may inflame, and, after establishing a communication with the bronchial tubes, may give rise to all the phenomena of gangrene of the lung.

2. *Into the Pericardium.*—This is, fortunately, a very rare direction, as the cases in which it has been noticed have been always fatal, either instantaneously by embarrassment of the heart's action, or within a few hours by acute pericarditis.[6]

3. *Into the Peritoneum.*—The tumour collapses, and violent and almost always fatal peritonitis is at once excited. This accident must not be confounded with the attacks of partial peritonitis which are so common before the tumour bursts in other directions. The rupture of the sac is often caused by external violence, in the form of a blow, fall, or strain. In the museum of St. Mary's Hospital is the calcified cyst of a hydatid, taken from the body of a man who dropped down dead after

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[1] See Cases XXXIX. and XL.; also Frerichs, Dis. of Liver (Syd. Soc. Ed.), ii. 235; Ogle, Path. Trans. xi. 299; Bristowe, Path. Trans. iii. 341; H. Davies, Path. Trans. i. 278; Davaine, Traité des Entozooaires, p. 437; into left lung, P. W. Latham, Lancet, Aug. 16, 1873.

[2] See Murchison, Ed. Med. Journ. Dec. 1865, Case XI., and case by Dr. Pollock, Path. Trans. v. 301.

[3] For examples, see Bright, Abdom. Tum. (Syd. Soc. Ed.), p. 49; Todd, Med. Times and Gazette, Jan. 5, 1854; Path. Trans. iv. 44; v. 303; viii. 92; ix. 28; Davaine, op. cit. p. 449.

[4] See cases by Peacock, Path. Trans. ii. 72; Pollock, ib. xvi. 155.

[5] Frerichs, op. cit. ii. 264; Peacock, Path. Trans. vol. xv. p. 247; Cayley, ib. xxvii. 171; Davaine, op. cit. p. 443.

[6] Two cases of rupture into the pericardium will be found in Davaine's work (p. 408); a third is recorded by Wunderlich (Med. Times and Gaz. Nov. 12, 1859, p. 488).

receiving a slight blow on the epigastrium from a comrade with whom he was sparring. The blow ruptured the cyst; the contents of the cyst escaped into the peritoneum, and the man died from shock. Many years ago Andral reported a case of hydatid of the liver terminating fatally by rupturing spontaneously into the peritoneum.[1] Three cases of fatal rupture in consequence of a fall are recorded by Mr. Cæsar Hawkins.[2] Three similar cases are mentioned by Frerichs; in two the rupture was caused by a fall, and in the third it was due to a strain; in one of the cases, death occurred within a quarter of an hour of the rupture. Eight additional cases have been collected by Davaine in which death ensued within a few hours or days of the rupture of a hydatid of the liver into the peritoneum; in several of the cases the rupture was caused by a fall or strain, and in one it occurred while the patient was wrestling with a comrade.[3] Rupture into the peritoneum was probably the cause of the fatal event in Case XLII. On the other hand, Bright records a case where what appeared to be a large hydatid tumour of the liver burst into the abdomen, without being followed by a fatal result.[4] Ogle also mentions the case of a patient who recovered after the symptoms of peritonitis resulting from the rupture of a hydatid cyst in the omentum.[5] Lastly, Dr. Fagge and Mr. Durham have found that, when needles were introduced into a hydatid of the liver, the fluid contents of the cyst seemed to ooze through into the peritoneum without any bad result.[6] These different results are, perhaps, due to the presence or absence of scolices and secondary cysts in the fluid which escapes, the entrance of the simple hydatid fluid into a serous cavity being, as Malgaigne has contended, harmless.[7] But, inasmuch as it is the exception for a hydatid to be barren, and there are no means of determining during life whether it be so or not, its rupture into the peritoneum must always be regarded with dread.

4. *Through the Abdominal Parietes or Lower Intercostal Spaces.*—This is not a common mode of termination, although several cases are on record. The contents of the hydatid may be discharged by an opening at the umbilicus or in some other part of the abdominal parietes, or in one of the lower intercostal spaces, and the patient may get well. Even here, however, the

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[1] Clin. Méd., Malad. de l'Abdomen, xliv. obs.

[2] Med.-Chir. Trans. vol. xviii. p. 124.

[3] Davaine. op. cit. p. 493.

[4] Abdom. Tumours, Syd. Soc. Ed. p. 47.

[5] Path. Trans. xi. p. 295.

[6] Med.-Chir. Trans. vol. liv., 1871.

[7] Traité de Méd. Opérat. 6me éd. p. 521.

cyst is apt to take on suppuration, and the patient may die from exhaustion or from peritonitis, or from extensive suppuration and sloughing of the abdominal parietes; or fatal hæmorrhage may occur from the interior of the sac, as in a case recorded by Dr. Bright. Of twelve cases where a spontaneous opening occurred, and of which I have collected notes, five at least terminated fatally, and in a sixth there remained, at the date of the report, a fistula discharging bile. Four also out of eleven cases observed by Finsen in Iceland were fatal.[1]

5. *Into the Stomach or Intestine.*—This is the most favourable direction in which the tumour can burst, although death sometimes results from the peritonitis which is set up around the opening, or from secondary abscesses of the liver,[2] and unfortunately it is not a common mode of termination. The tumour becomes flattened or disappears; and according as it opens into the stomach or the intestine, the hydatids are vomited or evacuated *per anum*;[3] sometimes they escape in both directions. The opening is usually small, so that the hydatids are discharged slowly.

Davaine has collected eleven cases where a hydatid tumour of the liver appeared to open into the stomach, of which six were fatal; and fifteen cases where there was reason to believe that it had opened into the intestine, of which only one was fatal. In one of Davaine's cases the tumour opened through the abdominal parietes, as well as into the stomach. In a case of large hydatid tumour of the liver which occurred in the Middlesex Hospital in 1859, under the care of my friend Dr. A. P. Stewart, where the liquid contents were drawn off by a trocar, the tumour subsequently burst into the bowel, discharging numerous cysts *per anum*, and the patient made a good recovery. In the 'Gazette des Hôpitaux' for 1850, a remarkable case is recorded where three hydatid cysts of the liver opened spontaneously, the first, in 1835, into the bronchi; the second, in 1845, into the stomach; and the third, in 1848, into the intestine: the patient recovered. Russell also has recorded the case of a man aged 36 who had two large

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[1] Budd, Dis. of Liver, 3rd. ed. p. 437; Frerichs, op. cit. ii. p. 237; Hawkins, Med.-Chir. Trans. xviii. pp. 153, 158; Bright, op. cit. p. 50; Griffiths, Lond. Med. Gaz. 1844, vol. xxxiv. p. 585; Davaine, op. cit. p. 384, Obs. V.; Ogier Ward, Path. Trans. iii. 100; Ransom, Brit. Med. Journ. 1873, ii. 376.

[2] See a case under Dr. Owen Rees, Med. Times and Gaz. June 20, 1857.

[3] For examples, see Frerichs, op. cit. ii. p. 237; Budd, op. cit. p. 452; Bright, op. cit. p. 49; Davaine, op. cit. p. 496.

hydatid tumours of the liver, one of which opened into the
pleura, and the other into the stomach and the bronchial
of the left lung.[1]

6. *Into the Urinary Passages.*—Although hydatid tur
of the abdomen or pelvis occasionally open into the ur
passages, echinococci and shreds of hydatid membrane
found in the urine,[2] I have met with no case where thi
happened when the primary cyst has been in the liver
1868 a case of this sort is said to have occurred in one o
London hospitals,[3] but it is not clear that the cyst was i
liver, or that it was a hydatid.

7. *Into the Biliary Passages.*—It is not uncommon
communication to be established between a hydatid tumo
the liver and one of the bile-ducts. In several cases
this has occurred, I have found the secondary cysts rupt
empty, and more or less stained with bile. The entran
bile, as was long ago stated by Cruveilhier, appears to be
to the life of the parasite, and in many cases probably co
tutes the commencement of a spontaneous cure, while in
cases it lights up severe and even fatal inflammatory actio
the cyst (Case XXXII.). Not only does bile enter the
but occasionally the contents of the cyst pass into the
ducts and gall-bladder, causing obstruction of these pass
with persistent and often fatal jaundice. In several insta
the passage of secondary cysts along the bile-ducts has
rise to all the symptoms produced by passing a gall-s
You have had opportunity of watching cases of this sort (C
XXXI. to XXXIV.), and several others will be foun
Davaine's work.[4] In one of the cases which have been u
your notice, the jaundice almost disappeared, although the s
remained colourless, in consequence of the bile draining
through the opening in the abdominal parietes (Case XXX
Mr. Hawkins has recorded a case where the common bile-
was obstructed by hydatids, without jaundice, owing to the

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[1] Med. Times and Gaz. 1873, i. 439.

[2] For several cases see Med. Times and Gaz. 1855, i. 159.

[3] Brit. Med. Journ. Nov. 7, 1868.

[4] Op. cit. p. 462. In rare instances a hydatid tumour appears to be develo
the bile-duct, although the possibility of such an occurrence is denied by D
Dr. Dickinson has recorded the case of a hydatid developed in the right hepati
where obstruction of the common duct was caused by a portion of the cyst, to
with inspissated bile (Path. Trans. xiii. 104).

escaping by a fistulous opening into a bronchus.[1] But now and then the biliary passages become sufficiently dilated to permit the evacuation of the contents of the cyst through them into the bowel. This is a rare occurrence, and most of the cases where it has been noticed have been fatal. A remarkable case is recorded by Dr. Hillier, where the contents of a hydatid tumour were discharged through the bile-duct into the bowel, but where the patient died in consequence of hæmorrhage from the wall of the cyst, the blood (derived apparently from branches of the hepatic artery) passing along the duct into the stomach and intestines.[2] Leudet reports a case of hydatid of the liver opening into a bile-duct; the patient died four weeks after the appearance of jaundice, and the extremities of the hepatic duct in the liver were found to be distended with pus.[3] Two cases are recorded by Dr. Wilks, where a hydatid cyst opened into a bile-duct, but where death was caused by peritonitis or by 'inflammation about the liver and ducts;' in one of the cases hydatid cysts had been vomited and passed from the bowel before the occurrence of inflammation.[4] Frerichs mentions a case where most of the contents of a hydatid had escaped by the bile-duct, but where the common duct ultimately became obstructed, and fatal rupture of the gall-bladder was the result.[5]

Case XXXIV. is an example of recovery after the discharge of the contents of a large hydatid cyst through the bile-duct into the bowel; but although the recovery appeared to be complete, several months afterwards the passage of some of the remaining contents of the tumour along the duct gave rise to severe pain and vomiting, and the muscular efforts in vomiting tore across some of the old adhesions: the result was fatal peritonitis.

In Sir Thomas Watson's case, already referred to (p. 59), the patient for eight or ten years, at intervals varying from ten to fourteen months in duration, had suffered a series of attacks precisely such as are commonly produced by the passage of a biliary concretion through the ducts of the liver. In May, 1847, just after one of these attacks, while searching for a gall-stone, he discovered two or three small hydatids in the fæces. In July he had the same symptoms for four or five days, and then vomited a hydatid as large as a pigeon's egg. This attack was followed by pulmonary symptoms, and in August he began to expectorate hydatids with large quantities of bile. The hydatids ceased to appear towards the end of November; the bile in the second week of February, 1848. After this he recovered; and twenty-three years afterwards he was alive and in good health, and in active practice as a medical man.[1] In a case referred to by Trousseau, the opening of the cyst into the bile-duct gave rise to biliary colic, which lasted for three weeks; upwards of three years afterwards there was a second attack of hepatic colic, followed by rupture of the cyst into the pleura and death.[2] Quite recently a very similar case has been observed by Dr. George Johnson,[3] but the patient died of acute peritonitis. The only other case of recovery, under like circumstances, which I have met with, is one referred to by Davaine, where there was reason to believe that a hydatid of the liver had ruptured into the gall-bladder, and where the patient recovered after a severe attack of biliary colic and jaundice, accompanied by the passage *per anum* of both hydatid cysts and gall-stones.[4]

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[1] Med.-Chir. Trans. xviii. p. 148.

[2] Path. Trans. vii. p. 222.

[3] Clin. méd., Paris, 1874, p. 412.

[4] Path. Trans. xi. p. 128.

[5] Op. cit. ii. p. 231.

8. *Into the Portal Vein.*—A hydatid of the liver occasionally opens into the portal vein or one of its branches. In a case where this had occurred, Leudet found numerous secondary abscesses in the liver.[5]

9. *Into the Vena Cava Inferior.*—In exceptional cases, a hydatid of the liver bursts into the inferior vena cava, and its contents, reaching the right side of the heart, become impacted in the pulmonary artery and cause instant death. Three cases of this sort are mentioned by Frerichs.[6]

But, independently of rupture, there are various ways in which a hydatid tumour may destroy life.

1. *By Marasmus and Exhaustion.*—This was the mode of death in Case XXXVIII., where a hydatid of the liver became so large that the entire abdomen was enormously distended by it, and respiration was seriously embarrassed. This case was further remarkable from the circumstance that there were dulness and fluctuation over the greater part of the front of the

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[1] Lectures 5th ed. 1871, ii. 631.

[2] Op. cit. iv. 285.

[3] Med. Times and Gaz., Jan. 1, 1876, p. 2.

[4] Op. cit. p. 477.

[5] Op. cit. p. 16.

[6] Op. cit. ii. p. 238. Two of these cases are related at greater length by Davaine (op. cit. p. 405).

distended abdomen, while the epigastrium and both flanks were tympanitic on percussion.

2. *By Pressure upon important Organs and Interference with their Functions.*—A hydatid tumour of the liver may compress the vena cava so as to cause anasarca and varices of the lower extremities,[1] or the portal vein, so as to induce ascites and necessitate recourse to paracentesis.[2]

By pressure upwards also it may rise as high as the second rib or the clavicle and greatly embarrass the respiration and the action of the heart; and by pressure on the stomach and intestines it may interfere with the function of assimilation, and cause various dyspeptic symptoms, emaciation and cachexia.

3. *By Suppuration or Gangrene of the Cyst, or Suppuration external to the Cyst, with or without Pyæmia and Secondary Purulent Deposits.*—Cases XXXII. XXXIII. XXXV. and XXXVI. afford illustrations of these modes of termination, and many similar cases are on record.[3] Bristowe has recorded a case where the secondary abscesses appeared due to obstruction of one of the ducts,[4] and in many cases pus has been found in the vein in the neighbourhood of the suppurating hydatid.

4. *By the Formation of Secondary Hydatid Tumours.*[5]—Secondary hydatid tumours may form in the liver or mesentery;[6] by a process of exogenous growth, such as happens more frequently in the hydatids which infest some of the lower animals. Cases have been observed in the human liver in which a secondary cyst budded from the outer surface of the parent hydatid;[7] and, if they be large or numerous, they may interfere with the patient's nutrition, and cause death by exhaustion, by peritonitis, or by uræmia from compression of the ureters, as in Case XLIII. Not uncommonly they form in the lung, and destroy life by inducing pneumonia. Cases XXVII. to XXIX. are examples of secondary hydatids of the peritoneum and

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[1] A case of this sort is recorded by Dr. Habershon, in Guy's Hospital Reports, 3rd ser. vol. vi. p. 182.

[2] See cases by Dr. Barker, Path. Trans. vol. vii. p. 225, and by Dr. Budd, Dis. of Liver, p. 451, and Hawkins in Med.-Chir. Trans. xviii. p. 149.

[3] For examples, see Bright, op. cit. p. 37; Budd, op. cit. p. 444; and Frerichs, op. cit. ii. p. 245.

[4] Path. Trans. vol. ix. p. 290.

[5] See cases recorded by Bright, op. cit. pp. 13, 23, and 30; Jones, Path. Trans. v. 298; Peacock, ib. 247 xv.; Gibb, ib. xvi. 157.

[6] This statement, which appeared in the first edition of these lectures, has been denied, but I have the authority of Dr. Cobbold for its correctness.

[7] Lect. on *Path. Anat.*, Wilks and Moxon, 2d. ed. p. 460.

mesentery; Case XXIX. was remarkable for the successful removal of the secondary cysts by Mr. Spencer Wells. Case XXIV. is an instance of a secondary hydatid tumour compressing the spinal cord, and causing paraplegia.[1] Dr. Barker relates the particulars of a case where death was due to the formation of a secondary hydatid in the brain.[2] An interesting case is recorded by Dr. Wilks, of a girl, aged nineteen, who died suddenly, having previously been in good health; a hydatid was found in the liver, and another at the apex of the left ventricle of the heart; the latter had burst, and discharged a loose hydatid into the cavity of the left ventricle.[3]

The treatment of hydatid tumours of the liver may be considered under the following heads.

1. Their *prophylaxis* is based on a knowledge of their cause. Hydatid tumours in man are developed from the eggs of a tapeworm which enter the body from without. This tape-worm, the *Tænia echinococcus*, the entire length of which does not exceed a quarter of an inch, inhabits the intestine of the dog and wolf, and is in no way connected with the pig, as is commonly believed to be the case. It has only four joints, and the ova are contained in the last, or proglottis, are voided with the fæces of the dog, and subsequently find their way into the human body with the food or drink. Arrived in the intestines, they are developed into embryos, which penetrate into the liver or other parts, in a way not yet satisfactorily explained, and are there developed into hydatid tumours.

But the ova of the *Tænia echinococcus* develope hydatids in other animals than man, and especially in the sheep. The hydatids of human beings, as Dr. Thudichum[4] observes, most frequently accompany them to their graves, or, at all events, they are not permitted to continue their dangerous existence, but the echinococci of sheep are again set free in the process of slaughtering, and are devoured by dogs, to be again developed into tape-worms. While, then, man does not contribute to the multiplication and propagation of echinococci, his constant liability to the disease is kept up by the cycle of infection which subsists between dogs and sheep.

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[1] Another case of a hydatid of the spinal column pressing on the cord is recorded by Dr. Ogle, Path. Trans. p. xi. 299.

[2] Path. Trans. x. p. 6.

[3] Path. Trans. xi. p. 71. See also Path. Trans. xv. p. 247. Cases of hydatid tumours of the heart, without any implication of the liver, are recorded by Habershon (Path. Trans. vi. p. 108), Budd (Path. Trans. x. p. 80), Peacock (Path. Trans. xxiv. p. 37), and Davaine, op. cit. p. 396. In Budd's case a hydatid tumour at the apex of the heart had burst, and loose hydatids were found in the right ventricle and in the pulmonary artery.

[4] Report on Parasitic Diseases in Quadrupeds used as Food, in Seventh Report of Med. Off. of Privy Council, London, 1865.

It follows, therefore, that for the prophylaxis of hydatid tumours in man it is necessary:—

a. To prevent dogs feeding on the offal of sheep and of other animals infested with hydatids. Dogs ought to be rigidly excluded from all slaughter-houses or knackeries, and 'dogs' meat' ought always to be thoroughly boiled.

b. To destroy, as far as possible, the tape-worms generated in the dog, for which purpose it would be well that all dogs were periodically physicked, and their excreta buried in the ground or burnt.

These are measures which are of national importance in such countries as Iceland, where the sheep-dog, during the long nights of winter, occupies the crowded dwelling of his master, and where hydatids are said to be the cause of one-seventh of the human mortality, and which merit attention even in our own country.

2. *Medicines.*—It must be confessed that little or no reliance can be placed on any medicinal agent for effecting a change in the size or in the structure of a hydatid tumour. Among the many remedies that have been proposed, common salt and iodide of potassium are the two which have been most relied on for destroying the life of a hydatid, but there is no evidence that either the one or the other is endowed with such a property. It is difficult to conceive how chloride of sodium can be unfavourable to the growth of a hydatid, when it is remembered how large a quantity of this salt is contained in the fluid contents of the cyst, and that, therefore, it must be compatible with, if not necessary to, the healthy existence of the parasite. And with regard to the preparations of iodine, there is not only no proof of their power to destroy the life of the parasite,[1] but

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[1] The following are references to instances in which iodide of potassium was thought to have effected the cure of a hydatid cyst:—Med. Times and Gaz. April 7, 1860. p. 344, and Oct. 19, 1872, p. 437; Lancet, Oct. 16, 1858; Brit. Med. Journ. 1871, i. 499. In one, at least, of the cases, the disappearance of the tumour appeared to be due to its having burst. The others may be viewed in connection with a case related by Dr. P. McGillivray, where a hydatid tumour, which it was intended to tap, disappeared spontaneously, a few days after the patient's admission into hospital (Austral. Med. Journ. Aug. 1865). As Dr. M. remarks: 'If the patient had been

piece of lint steeped in collodion, over which a compress and bandage are applied, and for forty-eight hours the patient ought to be kept in a recumbent posture, and every movement of the body be strictly prohibited: it may be well also to give an opiate at once, and, if there be the slightest pain, this may be repeated after a few hours.

One advantage of using a fine instrument is that it is unnecessary to wait for the formation of adhesions between the tumour and the abdominal wall, or to endeavour to induce them by measures not always free from danger before puncturing, or to leave the cannula in for twenty-four hours as practised by Jobert de Lamballe.[1] The walls of the cyst are so elastic that the small opening closes immediately that the instrument is withdrawn, and prevents subsequent oozing from the interior. If there be no adhesions, however, one precaution ought never to be neglected, viz. during the removal of the cannula to press the punctured portion of the abdominal wall against the cyst. By neglecting to do this the abdominal wall will be pulled away from the cyst in the extraction of the cannula, and the fluid in the cannula, perhaps containing scolices, may drop into the peritoneum.

The patient often experiences immediate relief from the sensation of tension and other unpleasant symptoms, from which he may previously have suffered, and within three or four days he is usually up and walking about. Not unfrequently an eruption of urticaria[2] is the source of some annoyance for the first day or two; in most cases the temperature is elevated from two to four degrees for several days; and more rarely the operation is followed by a feeling of uneasiness in the tumour, or by considerable pain and constitutional disturbance; but if the above rules be attended to, these symptoms soon pass off, and the patient makes a good recovery. It not unfrequently happens, however, that about a week or ten days after the operation the tumour again enlarges. This enlargement is not due to a re-accumulation of the hydatid fluid, but to inflammatory products thrown out between the collapsed parasite and the surrounding hepatic tissue, which are slowly re-absorbed. Under these circumstances it is well not to be hastily tempted to have recourse a second time to paracentesis. A certain degree of fulness may remain for many months, or even longer, in the site of the tumour, the existence of which has been cited as a proof that the operation has been unsuccessful. Yet inasmuch as the operation does not profess to remove the parent and secondary cysts, but only to kill the hydatid, and thereby avert those dangers which have been shown to result from its prolonged vitality, and to induce that slow process of atrophy which sometimes occurs independently of an operation, the fulness referred to is only what might be expected. If by the operation we can prevent the dangers likely to arise from a hydatid tumour, nothing more is necessary. Occasionally, however, the secondary enlargement of the cyst does not subside, and a fresh puncture becomes necessary. The fluid obtained by the second tapping has a higher specific gravity than normal hydatid fluid; it is no longer clear and free from albumen, and it always contains more or less pus. If the proportion of pus be small and the fluid not fetid, and if there be no severe constitutional symptoms, the first operation may be repeated with a reasonable hope of success; otherwise the case must be dealt with in the same way as an abscess, by making a free and permanent opening.

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[1] Trousseau, op. cit. iv. p. 294.

[2] This may be due to the escape of some of the liquid contents of the sac into the peritoneum, for the same symptom has been commonly observed when a hydatid has burst into the peritoneal cavity.

The safety and efficiency of the operation now recommended may be regarded as established. You have had many opportunities of satisfying yourselves on this matter in the cases under my care and that of my colleagues, during the last few years. In addition to the ten cases which I have from time to time brought under your notice (Cases XVI. to XXVI.), I would particularly direct your attention to two which were under the care of Dr. Greenhow, and which are reported in the eighteenth volume of the 'Pathological Transactions,' p. 127; in one of these the quantity of fluid drawn off amounted to 110, and in the other to 148 fluid ounces; five years after the operation in the former case, the patient was free from all signs or symptoms of the tumour. These and many other similar cases which might be quoted afford the best answer to the objection that the operation is only effectual where the tumour is small. It is true, that the operation, in killing the parasite, occasionally excites a certain amount of inflammation between it and the cavity of the liver in which it is embedded, but in most cases this, after a short time, spontaneously subsides, and it is only in exceptional cases that a second operation for the evacuation of pus becomes necessary. I have collected the particulars of 103 reported cases in which the operation was performed (see Tables at pp. 77–80). In 80 of the cases the operation appears to have been perfectly successful: in sixteen cases it was followed by suppuration, and a free opening was made into the sac, but all of the sixteen ultimately made a good recovery, and in several it appears to me that the necessity for a second operation was very doubtful. In seven of the cases (Table III.) the operation was followed by a fatal result; but in four, if not five, of the cases death was due to causes independent of the operation. In one of the remaining cases death was caused by sudden collapse twenty minutes after the puncture, and would probably have followed any operative interference; only about a drachm of fluid was drawn off; no fluid had escaped into the peritoneum, and there was no sign of peritonitis. In one case the patient died of peritonitis within twenty-four hours of the operation, but he was in a state of extreme prostration and emaciation before it was performed, and the propriety of having recourse to any operative procedure under the circumstances may be doubted. In estimating the results of the operation, those cases only ought to be taken into the calculation where it was resorted to as a curative measure, and those ought to be excluded where it was performed merely as a palliative, and where death was inevitable. I have therefore excluded from the Tables (appended to this lecture) several such cases, and others where the operation was performed with a large trocar, where caustic was employed to procure adhesions before puncturing, or where some irritating substance was injected after the withdrawal of the fluid, and also those where the hydatid had suppurated or been contaminated with bile before the operation, and where a different mode of procedure was advisable. The operation here recommended is only adapted for those cases where the fluid retains its natural limpid character, and the results of other operative procedures ought not to be confounded with it.

TABLE I.—*Cases of Hydatid of Liver in which the operation of puncture with a fine trocar and closing the orifice was followed by cure.*

No.	Authority	Sex	Age	Size of Tumour	Quantity of fluid in ounces removed	References and Remarks
1	Murchison	M	28	Moderate	5 & 20	Case 16, p. 87.
2	Do.	F	31	Do.	12	Case 17, p. 89.
3	Do.	F	6	Do.	14	Case 18, p. 92.
4	Do.	F	31	Large	20	Case 19, p. 93.
5	Do.	F	25	Do.	40	Case 20, p. 93.
6	Do.	F	60	Small	6	Case 21, p. 94.
7	Do.	M	8	Do.	6	Case 22, p. 94.
8	Do.	M	25	Moderate	8	Case 23, p. 95.
9	Do.	M	36	Large	16	Case 24, p. 96.
10	Do.	M	34	Multiple hydatid	4, 7, & 1½	Case 26, p. 99 Three distinct cysts were tapped.
11	Sir B. Brodie	M	12	Large	30	Med. Chir. Trans. vol. xviii.
12	Do.	F	20	Do.	60	Ib. p. 119. [p. 118.
13	Do.	M	14	Do.	60	Ib. p. 121.
14	Key	F	young	Do.	80	Bright on Abd. Tumours, Syd. Soc. ed. p. 42.
15	Boinet	F	19	Moderate	20	Traitement des Tum. hyd. du Foie, Paris, 1859, p. 13.
16	Do.	F	31	Small	4	Ib p. 14.
17	Do.	M	20	Moderate	20 & 15	Ib. p. 18. Two punctures were made at an interval of some
18	Demarquay	M	45	Do.	20	Ib. p. 30. [months.
19	Frerichs	M	46	Very large	120	Dis. of Liver, Syd. Soc. Ed.
20	Langenbeck	?	?	?	?	Ib. p. 269. [vol. ii. p. 268.
21	Do.	?	?	?	?	Ib. p. 269.
22	Récamier	F	young woman	Moderate	?	Rév. Méd. 1825, tom. i. p. 28.
23	Robert	M	?	?	?	Gaz. des Hôpitaux, 1857, p. 147
24	Do.	F	?	?	?	Ib. p. 147.
25	Cruveilhier	?	?	?	?	Ib. p. 147.
26	Richard	F	42	Large	40	Bull. Gén. de Thérap. 1855, p. 414. Two drachms of alcohol were injected.
27	Greenhow	M	25	Very large	21 & 110	Path. Trans. vol xviii. p. 127. Five years afterwards was still in perfect health.
28	Do.	F	30	Do.	148	Ib. p. 130. Dead six years afterwards from a return of the disease (another cyst.) Path. Trans. vol. xxv. p. 130.
29	Duffin	M	27	Moderate	28	Trans. Clin. Soc. vol. vi. p. 23.
30	Do.	F	26	Do.	21	Ib. p. 24.
31	Do.	F	39	Do.	28	Ib. p. 27.
32	Do.	M	61	Large	72	Ib p. 29. Fluid was partially
33	Do.	M	50	Do.	64	Ib. [purulent.
34	Church	F	23	Very large	120 & 114	Treatment of Hyd. Tum. of Liver, 1868, p. 15.
35	S. H. Ward	F	36	Large	37	Some affections of Liver; 1872, p. 59. More than a year afterwards another tumour appeared, which ultimately burst into stomach.
36	Brinton	F	19	Do.	30	Lancet, 1862, vol. ii. p. 639.
37	J. Hutchinson	F	30	Do.	30	Ib. 1862, vol. ii. p. 389.

TABLE I. (*continued*).

No.	Authority	Sex	Age	Size of Tumour	Quantity of fluid in ounces removed	References and Remarks
38	J. Hutchinson	F	33	Large	40	Brit. Med. Journ. Feb.20, 1864.
39	Do.	F	36	Do.	60	Ib.
40	W. Budd	M	35	Moderate	23	Ib. 1859, p. 275.
41	Fearn	M	30	Very large	85 & 40	Ib. Nov. 7, 1868. Second puncture, four months after first, brought away whey-like fluid.
42	Heaton	F	23	Large	40	Ib. Ap. 3, 1869.
43	Do.	F	20	Small	10	Ib. 1874, ii. 557. Aspirator was used and blood came at end of operation.
44	Sympson	M	29	Moderate	16	Ib. Ap. 30, 1870.
45	Southey and Savory	M	24	Very large	53	Ib. Aug. 6, 1870.
46	Ransom	F	20	Moderate	4¼	Ib. Sept. 28, 1872. For some days probable percolation
47	Do.	F	21	Large	13½	Ib. [into peritoneum.
48	Do.	F	25	Very large	33 & 72	Ib. Second tapping nearly six months after first. Fluid at first tapping contained bile and albumen.
49	Savory	?	?	?	?	Church. op. cit. 1868, p. 20.
50	Do.	?	?	?	?	Ib.
51	Phillipson	M	14	Moderate	23	Brit. Med. Journ. 1874, ii. 557.
52	Bradbury	F	23	Do.	16	Ib. 1874, ii. 558.
53	Do.	F	32	Do.	16	Ib. 1874, ii. 589.
54	G. Budd	M	25(?)	Very large	156	Med. Times and Gaz. May 19,
55	Holthouse	M	56	Do.	100	Ib. Jan. 6, 1855. [1860.
56	Sibson	F	33	Two Tumours	50 & ?	Lancet, July 18, 1868. Two tumours tapped in succession, at an interval of six
57	Anstie	F	6	Small	7	Ib. Aug. 13, 1870. [weeks.
58	Whittel	M	18	Do.	10	Ib. Oct. 15, 1870.
59	G. Hett	F	7	Moderate	14	Ib. Feb. 18, 1871. Doubtful if cyst in liver. Was punctured below umbilicus.
60	Scott Orr	M	20	Large	46 & 35	Glasgow Med. Jour. Jan. 1876.
61	McGillivray	M	56	Do.	30 & 20	Australian Med. Journal, Aug. 1865. Case iii. Second tapping two weeks after first.
62	Do.	M	27	?	?	Ib. Case vii.
63	Do.	M	45	Very large	180 & 100	Ib. Case xv. Second tapping six weeks after first brought away fluid tinged with bile.
64	Do.	F	23	Do.	114	Ib. March, 1867. Case xxiv.
65	Do.	M	5	Three cysts	20, 20, 10	Ib. Case xxvi. Three distinct cysts were tapped, none of which refilled.
66	Do.	M	6	Small	2	Ib. Case xxxvi.
67	Do.	F	11	Moderate	18	Ib. Case xxxvii.
68	Do.	M	17	Large	70	Ib. Case xxxviii.
69	Do.	M	51	?	?	Ib. July 1872. Case xl.
70	Do.	M	8	?	?	Ib. Case xli. [tapped twice.
71	Do.	F	28	?	?	Ib. Case xlvii.
72	Do.	F	30	?	?	Ib. Case lvi.
73	Do.	F	44	?	?	Ib. Case lviii.
74	Do.	M	59	?	?	Ib. Case lix.

TABLE I. (*continued*).

No.	Authority	Sex	Age	Size of Tumour	Quantity of fluid in ounces removed	References and Remarks
75	McGillivray	M	49	?	?	Australian Med. Journ. July 1872. Case lx.
76	Do.	M	32	?	?	Ib. Case lxvii.
77	Do.	M	3	?	?	Ib. Case lxx. Tapped twice.
78	Do.	F	13	?	?	Ib. Case lxxiii.
79	Bradbury	M	16	Large	22	Brit Med. Journ. Nov. 18, 1876.
80	Do.	M	36	Do.	40 & 30	Ib. Tapped twice.

TABLE II.—*Cases of Hydatid of Liver in which the operation of puncture with a fine trocar, and closing the orifice, was followed by suppuration of the Sac, a second free and permanent opening, and ultimate recovery.*

No.	Authority	Sex	Age	Size of Tumour	Quantity of fluid in ounces removed	References and Remarks
1	Garrod	F	19	Small	4	Lancet, Sept. 1, 1860.
2	Owen Rees	M	31	Large	38	Guy's Hosp. Reports, ser. ii. vol. vi. p. 17.
3	Boinet	F	8	Do.	40	Gaz. Hebdom. de Méd. sér. ii. 1864, i. p. 86. [p. 82.
4	Demarquay	M	50	Very large	160	Gaz. des Hôp. Fév. 19, 1859.
5	Babington and Cock	M	36	Large	10 & 80	Guy's Hosp. Reports, ser. iii. vol. vi. p. 179. The object of the operation was not to remove all the fluid at once, but by repeated punctures.
6	T. Sympson	F	39	Do.	60 & 30	Brit. Med. Jour. April 30, 1870. Second operation, five weeks after first, was probably unnecessary. On second occasion fluid partly purulent and tube was fixed in.
7	Bradbury	M	35	Do.	24 & 80	Ib. 1874, ii. 494. Second operation six weeks after first. Albuminuria before first puncture.
8	C. Brook	F	23	Moderate	6 & 12	Lancet, 1868, vol. i. p. 262. Second operation was probably unnecessary.
9	Duffin	M	32	[cyst Multiple	11	Trans. Clin. Soc. vol. vi. p. 31.
10	McGillivray	F	8	Moderate	20 & 20	Australian Med. Jour. Aug. 1865. Case xiv. Doubtful if the cyst which suppurated was that which had been first tapped. In first case cyst was close to surface; in second, matter was 3 inches from surface.
11	Do.	F	?	Do.	10	Ib. March 1867. Case xix.
12	Do.	F	12	Do.	30	Ib. Case xxxiii.
13	Do.	M	13	?	?	Ib. July 1872. Case xxxix.
14	Do.	M	61	?	?	Ib. Case xliii.
15	Do.	M	49	?	?	Ib. Case li. Tapped three times with fine trocar.
16	*Murchison*	F	32	Large	60	Case xxv. p. 97.

TABLE III.—*Cases of Hydatid of Liver in which the operation of puncture with a fine trocar, and closing the orifice, was followed by death.*

No.	Authority	Sex	Age	Quantity of fluid in ounces removed	References and Remarks
1	Moissenet	M	42	12	Arch. Gén. de Méd. Fév. 1859, p. 144. The patient was extremely prostrate before the operation, and died of peritonitis eighteen hours after.
2	Martineau	M	31	A few grammes	London Med. Record, June 23, 1875. Sudden collapse and death, twenty minutes after operation. Three days before, severe pain in epigastrium and r. hypochondrium. No sign of peritonitis at autopsy.
3	Dr. Scott Orr	F	18	38 Two cysts	Glasgow Med. Journ. Jan. 1876. Patient had cirrhosis, and for two months before operation deep jaundice, and shortly before operation severe pain in tumour. Eleven days after puncture fever set in and persisted till death, twenty-six days after puncture. Two cysts found after death—one in right lobe, which had been tapped, containing 3 pints of pus, and a second in left lobe containing 32½ oz. of turbid greenish fluid.
4	Bradbury	M	29	1 repeated	Brit. Med. Journ. 1874. ii. 525. Repeated punctures made with fine trocar. Only about an ounce of fluid drawn off each time. There were three large cysts, and death was due to one of them opening into lung.
5	Wiltshire	M	26	Large quantity	Lancet, Sept. 1, 1860. Liver contained three other cysts, each containing about one pint of fluid, besides the one that was punctured. Death appeared due to the pressure of the enormous liver upon neighbouring organs.
6	Murchison	F	21	60	Case xxvii. p. 100. There were multiple hydatids of liver and peritoneum, and death was due to suppuration of a cyst, distinct from that which was punctured.
7	Do.	M	45	28	Case xxviii. p. 102. There were multiple hydatids of liver and peritoneum, and ascites, &c., and death was quite independent of operation.

A careful consideration, then, of the whole matter—of the dangers of the disease when left alone, of the inutility of medicines on the one hand, and of the success hitherto obtained from a simple puncture on the other, leads to the practical conclusion that, in all cases where a hydatid tumour is large enough to be recognised during life and is increasing in size, it is advisable to puncture it at once. If the tumour appear to be diminishing in size, it may be well to wait; but it is unnecessary to wait for the formation of adhesions, or to endeavour to induce them. A hydatid tumour is not prone to form adhesions over its outer surface, like an abscess. By the time that adhesions form in the natural way, the tumour has attained a large size and is probably eating its way into some of the adjoining cavities; the chances are increased of its becoming inflamed and converted into an abscess; its walls also are much less elastic than at an earlier stage, and a puncture through them will close up less readily, so that there is a greater risk of fluid escaping into the peritoneum after removal of the cannula if there be no adhesions. While the walls are still elastic, the opening made by a fine trocar may be expected to close immediately that the instrument is withdrawn, and the existence of adhesions is therefore unnecessary.

4. *Evacuation of the Contents of the Cyst by a large Permanent Opening.*—In Case XXXII. you have had opportunities of studying the dangers to which a person must be subjected who has a large suppurating, or perhaps gangrenous, hydatid of the liver communicating by a free opening with the external atmosphere, and I have already pointed out to you that nearly one-half of the cases where an external opening forms spontaneously are fatal. The dangers are mainly four; viz. *a*. Exhaustion from the protracted discharge; *b*. Pyæmia and secondary inflammations; *c*. Hæmorrhage from the cavity in the liver; *d*. Peritonitis. Of 89 cases of which I have collected the particulars, where an opening of this sort occurred spontaneously (23 cases and 9 deaths) or was made by caustic, by a large trocar, or by incision, 28 were fatal, or the mortality was at the rate of 31·46 per cent. Many of those patients, also, who ultimately recovered, endured a protracted and exhausting illness.

When, however, the symptoms, or an exploratory puncture, show that the sac has undergone suppuration and that its contents are fetid, or that there are the constitutional symptoms of retained pus, a large permanent opening is the

only justifiable mode of operating, and the operation o
if possible, to be performed before the patient has be
exhausted and cachectic from fever and retained pus.
opening should be made with a large trocar, and a
cannula or india-rubber tube secured in the wound unti
whole of the hydatid contents have come away. The c
ought to be washed out in the first instance with a st
solution of chloride of zinc (20 grains to the ounce),
subsequently at least once a day with an aqueous soluti
carbolic acid (2 per cent.). In cases where the operati
followed by protracted suppuration, or when there is diffi
in keeping the pus free from fetor, it will be advisable to
a counter-opening and introduce a drainage-tube, in the
ner recommended by Boinet[1] and as commonly practise
empyema. Before operating in this way, it will alwa
well to ascertain the existence of adhesions, and, if nece
to produce them by an incision over the tumour plu
with lint, by the application of caustic potash, or by r
plied acupuncture with thirty or forty needles arranged
circle close to one another as practised by Trousseau ;
opening may be made by successive applications of c
potash, in the manner recommended by Récamier in ca
abscess.[2]

5. *Acupuncture.* A third plan of operating on hydatid tu
remains to be considered. In a communication made t
Royal Medical and Chirurgical Society of London on Nov
8, 1870, Dr. Hilton Fagge and Mr. Durham recorded
cases of hydatid of the liver treated by electrolysis, in
which the result was most satisfactory.[3] The operatio
sisted in passing two electrolytic needles into the cyst one
inches apart, both of which were connected with the ne
pole of a galvanic battery of ten cells. A moistened s
formed the termination of the positive pole, and this was
on the patient's skin at a little distance from the points
trance of the needles, and its position was changed from ti
time during the operation. The current was allowed to p
ten or twenty minutes. In several of the cases the operatio
followed by the signs of fluid in the pleura or peritone

[1] Gaz. méd. de Paris, 1860, No. 45.

[2] Frerichs, Dis. of Liver, Syd. Soc. Ed. ii. p. 148.

[3] Many years before, this operation had been tried successfully in
(Frerichs, op. cit. ii. 251).

that there was reason to suspect that the electrolysis acted as a kind of subcutaneous tapping, with effusion of the cyst fluid into a serous cavity, and this view was confirmed by the fact that in one case equally good results seemed to follow the introduction of needles into the cyst without the galvanic current. It is to be noted that the operation was in every instance free from danger; it was liable to be followed by some pyrexia and temporary refilling of the sac, but it did not set up active suppuration. Whether it be superior in this respect to puncture with a fine trocar has yet to be decided, but it certainly merits a further trial.[1]

Note.—The treatment of hydatid tumours advocated in the above lecture was recommended by me, in a memoir published in the 'Edinburgh Medical Journal' for December 1865, but has met with opposition from Dr. John Harley, of London, and from Dr. Finsen, of Copenhagen.

Dr. Harley,[1] who advocates the treatment of hydatid tumours of the liver by a large and permanent opening, gives a Table of '34 cases which were treated by a single puncture, evacuation of a portion or of the whole of the fluid, and immediate closure of the wound,' and states that, 'there were 11 *cures*, 13 *recoveries*, i.e. cases which were relieved by the operation, but which, since the tumour was not wholly removed, or the result sufficiently certified, cannot be regarded as radical cures, and 10 *deaths*.' Inasmuch as the parent and secondary cysts can never be 'wholly removed' by the operation of simple puncture, it is difficult to understand how Dr. Harley can admit that there was a 'radical cure' in any of the 34 cases. It is necessary, therefore, to explain that he seems to look upon the result as a *recovery*, and not a *cure*, if any trace of the tumour can *be felt* some time after the operation (as in my own case, No. 25 in his Table). The introduction of the 10 fatal cases into the Table, however, throws, in my opinion, an illegitimate discredit upon the operation in question, and it is, therefore, necessary to advert to them in detail.

Case 4.—In this case the tumour filled up the whole abdomen, and the operation of paracentesis (*with a large trocar*) was resorted to, with the object of relieving the impending asphyxia, and not as a curative measure. The patient, moreover, before the operation, was in a state of extreme marasmus and prostration, and the immediate cause of death was miliary tubercles in the lungs, and empyema. See Greenhow, 'Lancet,' 1862, ii. p. 476, and Murchison, 'Ed. Med. Journ.' Dec. 1865; also Case XXXVIII., p. 122 of this work.

Case 8.—There is no evidence that this case was fatal. Dr. Harley quotes the case from Mr. Cæsar Hawkins, and Mr. Hawkins from Dr.

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[1] *Med. Chir. Trans.* vol. xlix. 1866.

Thomas's 'Practice of Physic.' Mr. Hawkins observes, 'The result is not mentioned, so that it may probably be concluded that the case ended fatally,' but Dr. Thomas says nothing to warrant such a conclusion. 'Med. Chir. Trans.' vol. xviii. p. 121.

Case 9.—The operation was resorted to merely as a palliative measure: 8 pints of fluid were withdrawn from one cyst, and a second cyst, containing 12 pints, was found after death between the liver and the diaphragm. Dr. Abercrombie adds, 'The two cysts had so much injured the patient's constitution, that, although he was relieved by the operation, his strength quickly failed him.' Abercrombie, 'Dis. of Stomach,' p. 356.

Case 10.—In this case the opening was evidently a large one, and it is not stated whether it was closed up or not. But what is more important, the hydatid had suppurated before the operation. Hawkins in 'Med.-Chir. Trans.' vol. xviii. p. 157.

Case 11.—From the original account of this case in the 'Edin. Essays and Observ.' vol. ii. p. 299, it is clear that the boy was almost moribund at the time of the operation, and that, in addition to hydatids of the liver and spleen, he had ascites, general dropsy, and orthopnœa. It seems probable also that the peritoneum, and not the hydatid, was tapped.

Case 13.—In this case there was great constitutional disturbance, and the hydatid had suppurated before the operation. The patient also was pregnant and miscarried, and sank after this. Dr. Bright on 'Abdom. Tumours,' Syd. Soc. Ed. p. 41.

Case 15.—In this case there were two hydatid tumours. Three pints of fluid were drawn from one. This cyst did not again become enlarged, and the patient fancied herself cured, when death occurred from the rupture of the other cyst through the diaphragm into the lungs. Davaine, 'Traité des Entozoaires,' p. 447.

Case 16.—In this case the patient was in a state of extreme prostration before the operation. He was seized with syncope within five minutes, and died at the end of eighteen hours. Traces of recent peritonitis were found after death. The fatal result was no doubt determined in this case by the operation, but a large opening left patent is not likely to have led to a more favourable termination. Table III., No. 1, p. 80 of this work, and Archiv. Gén. de Méd. sér. v. tom. xiii. p. 145.

Case 19.—In this case the puncture was simply an exploratory one, preparatory to the application of caustic potash seven days afterwards. Death was due to tetanus twenty-five days after the puncture, and Récamier states, 'aucun accident n'a suivi la ponction.' Davaine, op. cit. p. 590.

Case 32.—In this case, according to Dr. Harley, no attempt was made to relieve the sac of its contents after the first puncture, and the hydatid fluid escaping into the peritoneum caused peritonitis and extension of the disease; but he omits to mention that the presence of a large and increasing amount of fluid in the peritoneum was diagnosed before the operation. Moreover, caustic potash was applied to the integuments before the cyst was tapped. Rogers in 'Brit. Med. Journ.' 1862, vol. i. p. 71.

It may seem surprising that as the data for my statistical Tables are in part derived from the same sources as Dr. Harley's, I should have been led to so different a conclusion. It is satisfactory therefore to me to find that Dr. Hilton Fagge and Mr. Durham have taken some pains to compare our Tables with the original data, and have entirely confirmed, in all essential particulars, the accuracy of my tabular statement.[1]

Dr. Finsen also advocates the operation of Récamier—viz. establishing adhesions by means of caustic, and then a free opening. I have not had the advantage of reading what he has written upon the subject, but I am informed by my friend Dr. Hjaltelin, of Iceland, that Dr. Finsen can only account for my success with the simple puncture, on the supposition that I have 'purposely concealed my unsuccessful cases.' In reply, I have only to state that all the cases in which I have been responsible for the operation are appended to this lecture, and that they will speak for themselves. How far Dr. Finsen is competent to designate the simple puncture of hydatid tumours a 'useless and dangerous operation,' I must leave to Dr. Hjaltelin, physician in chief, Reykjavik, Iceland, to decide.[2]

On the other hand, the success of the operation has been generally admitted by those who have had most opportunities of watching its effects. It is the treatment commonly practised in Iceland, where the disease is so common. The following passage from one of Dr. Hjaltelin's papers is worth quoting: 'I resolved myself to try the method of Récamier in some cases, which seemed to me more favourable for it than others, but am sorry to say that nearly one third of all those operated upon died. . . . . After I had quite given up the method of Récamier, and had returned to my old method of puncturing hydatid cysts, I happened to read Dr. Murchison's article "On Hydatid Tumours of the Liver, their Diagnosis and Treatment, 1865." As the experience of this physician is quite in accordance with my own, my faith in the treatment by puncture became strengthened, and I have since that time operated in a great number of cases with the best results.' Mr. Savory, of St. Bartholomew's Hospital, writes: 'The operation is much less likely to be followed by any untoward consequences than when a large trocar is employed. . . . . So convinced am I, from what I have hitherto seen, of the superiority of the fine trocar, that I would use it over and over again, in cases where the cyst refilled, before I would employ a large instrument.'[1] Mr. Durham, of Guy's Hospital, in the discussion upon his and Dr. Fagge's paper on the treatment of hydatid by electrolysis, stated that he had tapped eight cases by simple puncture with perfect success.[2] Dr. Duffin, of King's College Hospital, has recorded 7 cases of hydatid of the liver treated by simple puncture; all recovered, although in two the sac suppurated.[3] The treatment by puncture with a fine trocar and cannula was also strongly advocated by Dr. W. S. Church in his Oxford Graduation Essay published in 1868.[4] Lastly, in Australia, where the disease is very prevalent, the operation of puncture with a fine trocar is the treatment commonly adopted. Dr. McGillivray, among others, has pointed out the superiority of this plan to that of making a large and permanent opening. He has himself operated by the former method in 28 cases of hydatid of the liver, 24 of which made a good recovery, although in 6 the sac suppurated (see Tables I. and II). Four of the patients died; but in three of the four the operation was performed merely as a palliative, and the patients were previously the subjects of other maladies (disease of heart and dropsy, disease of lung and dropsy, and diphtheria) of which they died: in the fourth case the fluid drawn off by the primary puncture was 'brown bilious-looking stuff,' the sac suppurated, a large opening was made, and the patient died from gangrene of the liver.[5]

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[1] Med. Chir. Trans. 1871, vol. liv. p. 41.

[2] See papers by *Dr. Hjaltelin*, Brit. Med. Journ., Aug. 14, 1869, and Edin. Med. *Journ., Feb. 1870.*

The records of the following cases may serve to impress upon you more forcibly the symptoms and the dangers of hydatid tumours of the liver, and their appropriate treatment. In the first eleven cases (Cases XVI.–XXVI.) the cyst was punctured with a fine trocar, and after partial evacuation of the contents the opening was closed.

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[1] The Lancet, 1866, i. 524.

[2] See also Med. Chir. Trans. vol. liv. p. 40.

[3] Trans. Clin. Soc., 1873, vol. vi. p. 23.

[4] On the Treatment of Hydatid Tumours of the Liver.

[5] Australian Med. Journ., Aug. 1865; March 1867; and July 1872.

CASE XVI.—*Hydatid Tumour of the Liver—Paracentesis—Recovery.*

You have had an opportunity of studying the clinical characters of hydatid tumour of the liver, which have now been described, in the case of John N——, aged 28, who was admitted into Middlesex Hospital, under my care, on Dec. 3, 1866. He was a clerk, and had been in the Crimea for fourteen months, in 1855 and 1856. His previous health had always been good. In Sept. 1864 he had sore-throat and slight aching pain in his right side, and it was then discovered by Mr. Churton, of Erith, that he had a tumour in epigastrium, which was almost as large then as when he came under your notice. After that he suffered no uneasiness in tumour until Feb. 1866, when it became the seat of occasional darting pains, and on this account he was a patient in this hospital, under my care, from March 31 to April 18, 1866. Excepting these pains, which were very transient and unaccompanied by any tenderness, the patient's general health was good, and he had not the slightest fever. On April 7, an attempt was made to empty the cyst by means of a small trocar and cannula and an exhausting syringe, the puncture being made to the *left* of the middle line, where the tumour was most prominent. The action of the syringe, however, caused much pain in back and faintness, and the operation was abandoned after obtaining only four or five ounces of fluid, a quantity evidently much less than the tumour contained. Excepting an attack of urticaria, the operation was followed by no bad symptom.

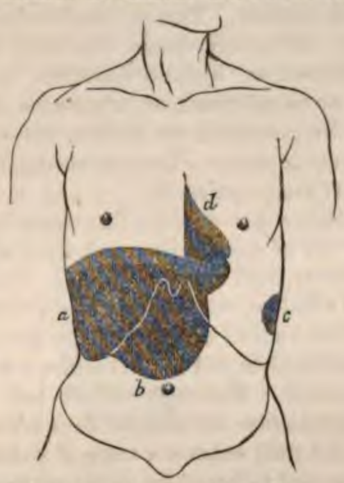

Fig. 12. Outline of Hepatic Dulness in case of John N——, at time of his admission into hospital, Dec. 3, 1866.

a, hepatic dulness; *b*, tumour; *c*, spleen; *d*, heart.

Patient was readmitted Dec. 3, partly on account of a return of the *slight pain from* which he had previously suffered, but mainly with object of having what was probably a second cyst emptied. At time of readmission, following note was taken of his state:—'Patient has a healthy appearance, and his only complaint is of a prominent tumour in epigastrium, extending into both hypochondria, and evidently connected with liver. It fills up space between sternum and umbilicus, and causes a slight bulging of ribs on both sides, particularly on the *right*. Its lower margin is about one inch above umbilicus. It measures about 6 inches transversely, and 5 inches from above downwards. Hepatic dulness is 6 inches in mesial line, and 5 inches in right mammary line; in right axillary and dorsal lines it is normal. These dimensions exactly correspond with those noted when patient left hospital last April. Upper margin of hepatic dulness is not more arched than natural. Tumour is globular, perfectly smooth, and not at all tender. It is very elastic, distinctly fluctuates, and presents the character known as 'hydatid vibration' in a marked degree It does not appear to be adherent, as its position varies with respiratory movements. No jaundice, no ascites, no enlargement of the spleen, and no albumen in urine. Tongue clean; bowels regular; no vomiting or pain after food; pulse 72.'

On Dec. 7, Mr. Moore introduced a fine trocar into most prominent part of tumour, to *right* of middle line, and drew off by cannula, without any syringe, twenty fluid ounces of fluid. This fluid was opalescent, colourless, and alkaline, with a specific gravity of 1009; it contained no albumen, but yielded a copious white precipitate with nitrate of silver; numerous hooklets and several entire echinococci were discovered with microscope. Although patient had been taking large doses of iodide of potassium for several days before both operations, on neither occasion did fluid contain a trace of iodine.

The operation was not followed by slightest febrile excitement or unfavourable symptom of any sort. On Dec. 12, patient got up, and on 18th he left hospital apparently well, tumour showing no tendency to enlarge, and hepatic dulness in right mammary line being only 3¾ inches.

On March 18, 1867, I again saw John N——, who informed me that four days after leaving he had been attacked with typhus fever, which he had probably contracted in hospital, and with which he had been dangerously ill. At commencement of the fever tumour appeared to enlarge, but by the time of his convalescence the swelling had quite subsided again, and now not the slightest trace of it can be discovered, vertical hepatic dulness in median line being only three inches.

March 9, 1868.—Patient presented himself at hospital, and was examined by Dr. H. Thompson, Dr. Greenhow, Mr. Moore, and a large number of students, but no trace of a tumour could be discovered.

CASE XVII.—*Hydatid Tumour of Liver, threatening to burst—Paracentesis—Recovery.*

On Aug. 3, 1864, Hannah S——, a very nervous woman, aged 31, consulted me about a tumour in region of liver. She was a cook in a medical man's family. In summer of 1863 she had been laid up for three weeks with a pain across stomach; but, with this exception, she had never suffered from any symptom of abdominal disease until about nine weeks before she came to me. She was then seized suddenly with acute pain in region of liver, which lasted about two hours. For several days she vomited everything she ate, and she had great pain in right side when she attempted to cough or to turn in bed. She kept her bed for a week, and did not resume her work for three weeks.

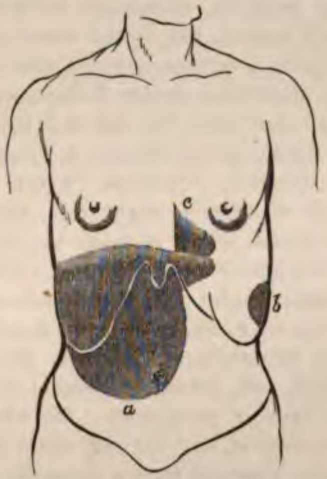

Fig. 13 represents the outline of Hepatic Dulness in the case of Hannah S——, in August 1864.

a, tumour; *b*, spleen; *c*, heart.

Liver was then first observed to be enlarged and prominent, but patient was unable to say whether this enlargement had existed before attack of pain or not. On examination, a slight bulging was found in right hypochondrium below ribs, this bulging being apparently continuous above with liver, extending to half an inch below umbilicus, and, transversely, from one inch to left of mesial line to about 3 in. to right. Vertical hepatic dulness two inches within right nipple was 7 in., 4¼ in. of the dull space being below edge of ribs. Tumour was tense, but elastic, and almost fluctuating. It was slightly tender on deep pressure. It did not appear to be adherent to abdominal wall. Posteriorly, hepatic dulness did not extend too high and upper margin not preternaturally arched. Respiratory sounds at right base were normal. Patient was slightly sallow, but had no decided jaundice. Tongue clean; appetite good; bowels regular. No ascites and no anasarca; urine contained neither albumen nor bile-pigment. Pulse 84.

On Aug. 7 patient had a return of pain in tumour, accompanied by vomiting and purging, lasting for two or three days. For several days after this attack tumour was tender, and over its surface coarse friction could be both heard and felt during respiratory movements.

On Aug. 19 Hannah S—— was admitted, under my care, into Middlesex Hospital, and placed on iodide of potassium, five grains three times a day.

On Aug. 24 tumour was noted as more tense and tender. On night of Sept. 2 patient had an attack of acute pain in right side, greatly increased by pressure, movement, or a long inspiration, and accompanied by much nausea, but by no vomiting or rigors. Pulse 96. Under use of opium, poultices, and rest, these symptoms gradually subsided, but tumour continued tender, friction was again distinguishable for several days, and pulse did not fall below 96. On Sept. 9 patient had another similar attack of pain, but more severe; pulse rose to 116, and friction returned. On Sept. 14 pain was less, but tumour was observed to extend more to right side, and was less rounded. On Sept. 17 another severe attack of pain; and indeed, since Aug. 24, tumour had never been free from tenderness, while patient felt herself gradually getting weaker, pulse being rarely below 108.

Although there was no evidence of firm adhesions over tumour, it was now determined to puncture it. From first, tumour had been diagnosed as a hydatid, and indeed the object of patient's admission into hospital was to have it punctured. All who examined it were agreed that it contained fluid, and the only other affections at all likely to produce appearances observed were a distended gall-bladder and an abscess of liver. The tumour did not occupy quite the situation, and latterly did not present shape of a distended gall-bladder, and there had never been jaundice. The persistent pain and tenderness noted for several weeks pointed rather to abscess, but there had been no rigors or perspirations, and, moreover, the tumour had not increased much in size since it had been first observed. Supposing the tumour to be hydatid there was reason to fear that it was about to burst.

On Sept. 20 Mr. Hulke tapped tumour with a fine trocar, the cannula of which was scarcely so large as a No. 1 catheter, and drew off about twelve fluid ounces of clear limpid fluid, specific gravity of which was 1009. No echinococci or hooklets could be discovered in it, but it was found to contain a large amount of chloride of sodium and no albumen. It did not contain a trace of iodine, although iodide of potassium had been taken almost continuously for several weeks.

In removing cannula, abdominal parietes were pressed down against tumour, and puncture was afterwards covered with collodion and a pad. Patient was kept on her back for forty-eight hours, and not permitted to move. Twenty drops of laudanum were administered immediately after operation, and for two days an opiate was given about once in four or six hours.

The night after the operation, patient slept well. On following day, urine was retained, and was drawn off by catheter; and on Sept. 22 abdomen was distended and tympanitic, skin hot and dry (temperature 101°), pulse 120, and much thirst. Still there was much less pain and tenderness over tumour than before operation. Bowels had not been open for two days. An enema of turpentine and confection of rue brought away a large quantity of flatus, and patient at once began to improve. On Sept. 26, pulse 96, tongue clean and moist, and appetite returning. For first time for several weeks, patient could tolerate free manipulation of tumour, dimensions of which were much reduced. On Sept. 27, pulse 84; collodion was removed from wound, from which not a drop of discharge had escaped. On Sept. 30 patient was able to get up. Convalescence was retarded by an attack of facial neuralgia and other trifling ailments; but on Nov. 22, patient was able to leave hospital. Dimensions of tumour were gradually diminishing, so that dulness from upper margin of liver to lower margin of tumour did not exceed 5¾ inches. Tumour also was quite soft and free from tension, and could be manipulated without causing pain. Tongue clean and moist; appetite and digestion good. Pulse 100.

June 1867.—Nearly three years have now elapsed since operation, and during most of that time patient has been able to follow her occupation as a cook, subject only to flatulence and other symptoms of dyspepsia and hysteria. Only a slight fulness is now perceptible in epigastrium.

Early in 1868, patient wrote that she was quite well and was about to be married. In autumn of that year she had a child, who died soon after birth. After this she fell into low spirits, and she was again under my care in Middlesex Hosp. during Jan. 1868. She was then suffering from dyspepsia, flatulence, and hysterical pains. A hard mass about size of an orange could still be felt in site of tumour; it was quite painless, and did not seem to be connected with patient's symptoms.

July 1873.—Patient wrote to say that she was much in same condition and that tumour was no larger.

Case XVIII. was remarkable for the early age of the patient.[1] Trousseau has recorded a case where the patient was also only six, and adds that Davaine, in his great work on Entozoa, had not been able to collect more than 14 cases in subjects under fifteen years of age; but in one of Davaine's cases which he quotes from Cruveilhier, the subject was a child only *twelve days* old, and the cyst had already opened into the descending colon. (See also Case XXII. and Tables I. and II.)

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[1] Clin. Med., Syd. Soc. Trans. iv. 264.

Case XVIII.—*Hydatid Tumour of Liver—Puncture with fine Trocar—Recovery.*

Elizabeth C——, aged 6, adm. into Middlesex Hosp. under my care Dec. 3, 1867. With exception of whooping-cough at age of 3, she had always enjoyed excellent health; but her mother, almost since she was an infant, had noticed that she was larger about the waist than natural. Three months before, the girl had been seen by Miss Garrett, M.D., who diagnosed hydatid of liver. Since then mother thinks that tumour has been increasing, but the only uneasiness child has experienced has been an occasional feeling of sickness, a morning cough, and slight pain in region of liver. On admission, patient was a robust, healthy-looking child, who seemed to have nothing amiss with her, with exception of a swelling in epigastrium, extending vertically from lower end of sternum to umbilicus, and 2½ inches laterally to either side of mesial line. The tumour was globular, smooth, painless on manipulation, and with distinct fluctuation, and 'hydatid vibration.' It was quite movable over subjacent parts, and did not appear to be adherent to abdominal parietes, as it descended readily with inspiration. Although evidently connected with liver, area of hepatic dulness was not generally increased, its extent in right mammary line measuring only 2½ inches. Girth of abdomen over tumour was as follows:

	Dec. 3.	Dec. 20.	Jan. 16.	Jan. 24.	March 9.
At umbilicus . . .	24·3	23·3	24·5	22·75	22·5
At ensiform cartilage . .	24·5	23·5	23·5	23·75	23·5
Half-way between umbilicus and ensiform cartilage .	25·75	24·66	25·25	24·	22·5

Tongue clean, appetite good, bowels regular. There was neither ascites nor jaundice. Pulse 96. She was ordered a draught containing two grains of iodide of potassium three times a day.

On Dec. 10 Mr. Hulke punctured tumour with a fine trocar, and drew off fourteen fluid ounces of fluid. This was colourless, slightly opalescent, with a specific gravity of 1010, and contained no albumen, but a large quantity of chlorides; neither echinococci, nor hooklets, nor any trace of iodine could be detected in it. Two hours after operation patient was sitting up in bed laughing and talking as if nothing had happened. During following night, however, she had several attacks of vomiting (which was, perhaps, the effect of chloroform that had been administered), and for two days the pulse rose to 140, and the temperature was as high as 100·8°; but there was no tenderness of abdomen, nor thoracic breathing.

On Dec. 13 temperature and pulse were again normal, and after this patient had no bad symptom, except that from Dec. 20 till Jan. 14 tumour appeared to increase again slowly in size, so that the question of performing paracentesis a second time was entertained. This, however, was abandoned, for tumour began to diminish spontaneously, as will appear from table of measurements. On March 9 there was no perceptible bulging and scarcely any tumour to be felt.

CASE XIX.—*Hydatid Tumour of Left Lobe of Liver—Paracentesis—Recovery.*

Emma H——, aged 31, adm. into Middlesex Hosp. Dec. 4, 1868. Married and had five children; youngest child born sixteen months before had survived birth only three days. After this suffered from languor, prostration, and low spirits, and while in this state attention was first drawn by a feeling of heat to a swelling in left hypochondrium, which, however, had not materially increased in size since it had been first noticed; nor had it prevented her following her ordinary household occupations. On admission, there was a tumour filling epigastrium, extending to 2½ in. below umbilicus, measuring 7½ in. vertically, and 10 in. transversely, bulging forwards, tense, smooth, fluctuating, with distinct 'hydatid vibration,' and slightly tender. The tumour evidently grew downwards from liver, which did not extend too high upwards; it did not appear to be adherent to abdominal wall. The patient was anæmic, but her general health was in other respects good. Dec. 6, ordered 5 grains of iodide of potassium three times a day. Dec. 10, paracentesis with fine trocar: one pint of fluid drawn off, limpid, sp. gr. 1009, and containing much chlorides, but not a trace of albumen or of iodine; last few ounces had a sp. gr. of 1012 and contained blood and bile-pigment. No bad symptom followed operation; pulse never exceeded 80, and skin was cool. On Dec. 18 patient got up, and on 28th she left hospital.

July 19, 1872. Patient called at my house. Has had two children since operation, and is now suckling second, aged 12 months. A small hard, non-elastic, painless tumour can still be felt in epigastrium, but this is the source of no inconvenience.

CASE XX.—*Hydatid of Left Lobe of Liver—Paracentesis—Recovery.*

Mrs. R——, aged 25, consulted me on Dec. 29, 1871, on account of a smooth painless tumour in hypochondrium, apparently growing from left lobe of liver, and reaching down to umbilicus, which had been first noticed two years before and which had since slowly increased. Girth over most prominent part of tumour 29¼ in.: right side, 14 in.; left, 15¼. Chief complaint was of constant pain in back, and atonic dyspepsia. Improvement took place under use of nitro-muriatic acid and strychnia; and on *March* 5, 1873, patient was stouter and stronger, but tumour was larger; girth on left side over tumour 16 in.; right side, 14 in. *March* 24. Paracentesis with fine trocar. Drew off 40 fluid ounces of limpid fluid, containing much chlorides but no albumen;

sp. gr. 1010. *March* 25, no pain; pulse 74; temp. 98·4°. *March* 27, pulse 96; temp. 102·5°. *March* 31. Up and going about, but tumour appears to be slightly larger again; pulse 84; temp. 101·5°. *April* 7. Tumour smaller again, and has got on stays first time for years. Less pain in back than she has had for a long time. Pulse 90; temp. 101°. *May* 7. Much better, and gained flesh. Girth equal on two sides; viz. 14½ in.

July 1875. Patient is in enjoyment of excellent health, and there is no sign of tumour.

CASE XXI.—*Hydatid of Right Lobe of Liver—Neuralgic Pain—Paracentesis—Recovery.*

On Oct. 30, 1873, I saw in consultation with Mr. R. Phillips, of Leinster Square, a lady, Mrs. M——, aged sixty, who had a large smooth tumour in right hypochondrium, connected with liver. Hepatic dulness in front arched up to nipple, measured 8 in. in right nipple line, but did not ascend too high at back. Lower margin of right lobe descended to level of umbilicus. Lower right ribs and cartilages formed a visible bulging forwards; girth over most prominent part from spine to middle line in front 17 in., and at corresponding part of left side 15¼ in. Over most prominent part of swelling between ribs, as well as below them, decided elasticity and even obscure fluctuation; no tenderness. Tumour had been first observed ten months before, and had not materially increased. Ever since she had been liable to severe neuralgic pains, and a disagreeable feeling of tightness about liver; and four months after she became aware of tumour she had an attack of pleurisy on right side with effusion, which had been absorbed. Her only other symptoms were some nausea and loss of appetite.

I advised a puncture with a fine trocar between the ribs, and a few days afterwards Mr. Phillips drew off from swelling by aspirator six ounces of fluid. This was faintly opalescent, had sp. gr. of 1010, was unchanged by boiling, but became slightly opaque on adding nitric acid, formed a dense white deposit with nitrate of silver, and contained numerous echinococci. The tightness and neuralgic pains were at once relieved, and lower margin of liver receded almost to margin of ribs. No bad symptom followed, and in July 1875 patient was in good health, and had no sign of tumour. Oct. 1876. Still in good health. No pain, or swelling.

CASE XXII.—*Hydatid Tumour bulging from upper surface of Liver, and pressing it down—Paracentesis—Recovery.*

Albert D——, aged 8, a pale rather thin boy, was brought to St. Thomas's Hosp. Jan. 1, 1874, on account of a swelling in upper part of abdomen, which had been first noticed between two and three years before, and which had slowly increased in size, without pain or other uneasiness. The swelling extended from ribs to 1½ in. below umbilicus. Its surface was marked by a transverse furrow, 3 in. above umbilicus. Below this its consistence was firm, and what was felt appeared to be the liver; while between furrow and ribs was a globular prominence, smooth, painless, fluctuating, and yielding distinct 'hydatid vibration' on percussion. There was dulness over right lower ribs, rising to about level of normal hepatic dulness, but its upper margin was too much arched. Hepatic dulness in r. m. l., including liver, 9½ in.; girth of abdomen round most prominent part of tumour 25 in.; from ensiform cartilage to umbilicus 7 in.; from umbilicus to pubes 4½ in. Pulse 84; apex of heart elevated, beating between third and fourth ribs. Tongue clean; appetite good; bowels regular; no jaundice; no pain; no ascites or œdema of legs.

Ordered Fer. et quin. cit. gr. iij t. d. s.

Jan. 8. Paracentesis at 10 A.M. with fine trocar; 6 ounces of clear fluid drawn off, containing much chlorides, but no albumen; sp. gr. 1011. The operation was followed by no pain or uneasiness, but temperature same evening rose to 102·8°, and on the three successive nights it was 103·1°, 101·8°, and 101·5°. On morning of 9th it was 101·2°, but on other mornings it was normal. During night of 9th patient was somewhat restless and thirsty, but by 12th fever had subsided, and when patient left hospital on 22nd girth over most prominent part of tumour was 24¼ in., and swelling was much less prominent and tense, but to right of cyst which had been tapped appeared to be a second, in which, however, fluctuation was not very distinct. This was not interfered with.

CASE XXIII.—*Hydatid of Liver commencing to suppurate—Paracentesis —Recovery.*

On March 11, 1876, I saw, in consultation with Dr. Barker, of Hornsey, Mr. P——, aged 25, who had a tumour, presenting all characters of hydatid, projecting downwards from right lobe of liver. It formed a distinct prominence, which measured 7½ in. both vertically and transversely. It had been discovered about previous Christmas, when it first became seat of slight pain. Since first noticed it had increased unmistakably, but not greatly. I tapped it with a fine trocar, and drew off eight ounces of thin fluid, which was turbid, of 1010 sp. gr., contained much chlorides and a little albumen, and threw down a creamy deposit made up of pus, oil, cholesterin, hooklets, and shreds of hydatid membrane.

April 25.—For three or four days after puncture, much pain and sickness, but all subsided under opium and effervescing draughts. At end of two weeks tumour much smaller, but in last fortnight has been enlarging again, and now measures 6 in. vertically and 7½ in. transversely. General health good.

June 8.—Gained flesh and colour, and tumour much smaller, measures 4½ in. vertically, and 5½ in. transversely.

Oct. 3.—Much stouter, and general health excellent. Has no discomfort from tumour, which continues to get smaller, and feels much harder.

CASE XXIV.—*Hydatid of Liver—Paracentesis—Recovery.*

Deacon B., aged 36, railway station-master, adm. into St. Thomas's Hosp. June 1, 1876. Tertian ague at 18, in Cambridgeshire. Excepting this, health had been good. For eight years had complained of a feeling of weight and occasional slight pain in region of liver; six months ago these symptoms attracted more attention, and three months ago first noticed a swelling which had slowly increased in size. Six weeks ago had an attack of acute pain in tumour, subsiding in 24 hours, but leaving him weak, so that he kept his bed for a fortnight. Lost about a stone in weight, but regained it before admission.

On admission, a distinct prominence between costal cartilages and umbilicus, more on right side than left, evidently due to a growth from liver, smooth, rounded, painless, tense but elastic; no thrill or vibration; its lower margin reaching to umbilicus. Liver generally not enlarged; upper margin not too high; dulness in r. m. l. 7 in., in mesial line 9 in.; girth of abdomen over tumour 36¼ in. Body well nourished; no pyrexia or perspiration; appetite good, and sleeps well. Chief inconvenience from swelling is some dyspnœa on exertion, and a feeling of tightness after food or when he stoops.

June 5.—Paracentesis with fine trocar; 16 ounces of clear fluid drawn off; sp. gr. 1009: abundant chlorides; not a trace of albumen, even with cold nitric acid test.

June 17.—Got up after two days, and has not had a bad symptom. Temp. on night of June 7 rose to 100·8°, but with this exception has been normal throughout. No pain; no urticaria. No evidence of cyst refilling; lower margin 2½ in. above umbilicus; girth over its most prominent part 35 in., same as day after tapping.

July 15.—Came to show himself at hospital. Has had flatulence and uneasiness about tumour, but this has not increased in size; girth over it still 35 in., but patient has got stouter.

Oct. 20.—Examined him again. Tumour imperceptible, and excepting flatulent dyspepsia general health good. Has followed employment for last three months.

In Case XXV. the operation of simple puncture was followed by suppuration of the sac, with much fever, and it was necessary to make a free opening. It is to be noted, however, that before the operation the patient had symptoms of congestion of the liver, and that the immediate cause of the severe inflammation of the sac was a chill. The case further illustrates the good effects of antiseptic treatment in dealing with a large abscess of the liver, full of fetid pus.

CASE XXV.—*Hydatid of Liver—Paracentesis—Suppuration—Free Opening—Recovery.*

Hannah B——, aged 32, adm. into Middlesex Hosp. Nov. 30, 1869. In Nov. 1866 began to suffer from occasional pain, not severe, in right side. In Nov. 1868 first noticed a fulness in right side, which has continued to increase. One month before admission lost appetite, began to suffer from nausea, vomited bile occasionally, and became slightly jaundiced (hepatic congestion). On admission, liver greatly enlarged, extending in front from upper border of fourth rib to 2 in. below umbilicus; measurement in r. m. l. 12 in. and in mesial line 11 in. Posteriorly, upper margin of hepatic dulness not higher than natural. Girth over most prominent part of tumour one inch below lower end of sternum, right side 16¼ in., left 15¼ in. Surface of tumour below ribs smooth, elastic, painless, except close to ribs, where it is slightly tender and distinctly fluctuating, but no 'vibration.' Slight jaundice. Urine contains same bile-pigment, but no albumen; motions contain bile.

Dec. 9.—Jaundice and dyspeptic symptoms have almost disappeared, but urine still contains bile. Cyst tapped with fine trocar, and 60 ounces of alkaline limpid fluid drawn off, containing much chlorides, but no albumen; sp. gr. 1009: no echinococci or hooklets found. On evening of same day felt chilly; pulse 102; temp. 102·2°: but no pain. After 24 hours the febrile symptoms subsided, and patient felt more comfortable than before operation, but the urine still contained bile. On *Dec.* 16 she got up.

Dec. 22.—On evening of Dec. 20 had headache, but yesterday was up all day and went down to Board Room to pass for discharge, when she fancied that she caught cold. Last night she began to suffer from thirst, and to-day pulse 120: temp. 103·8°. No rigors, but felt chilly this morning; no abdominal pain. Next night she had no sleep, perspired profusely, and had frequent retching. On Dec. 23 she had slight pain in region of tumour on taking a deep breath; pulse 118; temp. 101·4°. In the evening she had a severe rigor followed by perspirations.

Dec. 24.—Jaundice increased. Urine contains a trace of albumen and much lithates. Pulse 110: temp. 104·4°.

Jan. 1.—Still very ill. Pulse has varied from 106 to 120, and temp. from 101·2° to 105°. No return of rigors, but perspires freely at night. Urine always contains albumen ($\frac{1}{20}$). Occasional retching and jaundice continue. For several days has had frequent cough, and to-day there are coarse moist sounds over lower two-thirds of both lungs at back, and sibilant râles in front. Tumour is evidently enlarging again. Sleeps little.

Jan. 10.—Early this morning had a second slight rigor followed by perspiration, but is on the whole better. Temperature for several days has been falling and is now normal. Less jaundice. Urine still contains albumen. Still much congestion of lungs.

Jan. 26.—General condition much improved. Temp. 98°, and has rarely exceeded 100°. Cough much less, breathing easier; very few râles in lungs. No albumen in urine. For two days has had rather severe pain in tumour, which continues to enlarge.

Feb. 2.—Pain continues, and pyrexia has increased, temp. varying from 98° to 101·8°. Pulmonary congestion increased. Girth half-way between sternum and umbilicus is now 37 in. No albumen in urine. About a pint of thin fetid pus was drawn off from tumour by a small trocar, and a piece of Vienna paste about size of shilling was on Feb. 3 applied at spot where puncture had been made.

Feb. 6.—An incision was made into eschar produced by paste, a large trocar was thrust in, and 90 ounces of fetid pus containing numerous large shreds of hydatid membrane were drawn off. The cavity was washed out with a solution of chloride of zinc (gr. x ad ℥j) until the liquid returned almost clear; the opening was covered with lint soaked in carbolic oil, outside which was placed a quantity of carded oakum.

Feb. 8.—Much better. Pain much relieved. Temp. normal. Since *Feb.* 4 urine has again contained albumen ($\frac{1}{10}$ to $\frac{1}{20}$). Girth at level of opening 31 in.

After a few days the opening became choked up, and the general symptoms became worse. The congestion of the lungs increased and there was great dyspnœa. On *Feb.* 17, 60 ounces of pus (not fetid), with hydatid membranes were let out by opening, into which a perforated drainage tube was fastened, and cavity was again washed out with solution of chloride of zinc. Pus without any fetor was discharged in large quantity by the tube. The general symptoms slowly improved. On *Feb.* 18 albumen, and on *Feb.* 21 bile-pigment disappeared from urine, but both returned on *March* 2 for a few days, during which the discharge was occasionally fetid until the cavity was again washed out with chloride of zinc. The patient, however, still suffered much from cough and dyspnœa, and perspirations at night, and from *March* 8 to 15 she had frequent retching. Between *March* 14 and 17 enormous masses of thick tough hydatid membrane (parent cyst), came away while the cavity was being washed out, and after this there was a rapid improvement. On *March* 24 the discharge had almost ceased. On *April* 1 patient got up. On *May* 6 tube was removed, and on *June* 6 she left hospital with wound almost cicatrised. The urine was free from albumen; lungs sound; and daily gaining flesh and strength. Lower edge of liver felt 2 in. above umbilicus; hepatic dulness in r. m. l. 5 in., and girth over wound 29 in.

In spring of 1875 Hannah B. was free from all sign of her former disease, but she had become enormously fat, and was a confirmed spirit-drinker.

In the following case there were numerous hydatid cysts in the liver. Three of these were tapped with success, but there was reason to suspect the existence of a larger cyst deeply seated. It is to be regretted that the patient left the hospital before an attempt was made to reach this by an exploratory puncture.

Case XXVI.—*Multiple Hydatid Tumours of Liver—Jaundice and Diarrhœa—Paracentesis of three Cysts.*

Henry A——, aged 34, labourer, adm. into Middlesex Hosp. Feb. 16, 1869. Five years before had sustained an injury of right side, from wheel of a waggon pressing against it, but he had not suffered much inconvenience from this, and had enjoyed good health till one day in Aug. 1868, while unloading a van in the sun, he fell and was unconscious for three minutes, after which he was laid up for three weeks with vomiting and diarrhœa and light drab-coloured motions, but without jaundice, headache, or giddiness. After this he returned to work, but suffered from flatulence, pain in stomach after food, and occasional pricking pains in situation of liver. One month before admission he was again seized with violent diarrhœa, this time accompanied with jaundice, but without vomiting. About same time he first noticed a swelling in right side, and a feeling of weight, which was much increased whenever he lay on left side. The jaundice had increased in intensity up to time of admission, and in five weeks he had lost 7 lbs. in weight.

On admission, patient was fairly nourished, and with exception of being rather deeply jaundiced, did not appear ill. Liver greatly enlarged, its dulness in r. m. l. extending from ½ in. above nipple to 4 in. below ribs, and measuring 10½ in. Corresponding to gall bladder was a rounded projection, about size of an orange and distinctly fluctuating; and fluctuation could also be made out to right of this below edge of ribs, the two fluctuating spaces being separated from one another by a depression in which no fluctuation could be felt. Lower margin of liver could be felt below seat of fluctuation, hard and sharp. Posteriorly hepatic dulness did not ascend too high, but fluctuation was tolerably distinct between tenth and eleventh ribs. Veins of abdominal parietes unusually distinct, and splenic dulness increased; no ascites; diarrhœa persisting, five motions on morning of admission; motions clay-coloured and devoid of bile. Much flatulent distension of abdomen. Appetite good. Urine 1024, free from albumen, but loaded with bile-pigment. Pulse 40, regular; heart displaced upwards; no bellows-murmur. Dyspnœa on exertion and slight cough, but physical signs of lungs normal.

Feb. 25.—Pulse 72. Diarrhœa abated. Under ether spray a fine trocar was introduced into anterior fluctuating space, and about 4 ounces of hydatid fluid drawn off; clear, sp. gr. 1011, and containing no albumen, but abundance of chlorides. A wire could only be passed in two inches through cannula, and draining away of the liquid made no difference in tension of fluctuating swelling to the right.

March 5.—Pulse has varied from 56 to 72, and temp. has been normal, but diarrhœa has returned. A puncture was made into fluctuating space to right of first, and 7 ounces of fluid let out; clear, sp. gr. 1009, and containing no albumen, but much chlorides.

March 19.—Pulse has varied from 56 to 72, and temp. has been normal. Diarrhœa and jaundice continue, and motions contain no bile. A rounded elastic tumour about size of a turkey's egg can be felt in left groin, which patient first observed about a fortnight ago. Since *March* 10 there has been slight increase of fulness in situation of cyst first tapped, but no tenderness or fluctuation. Urine free from albumen.

April 2.—Slight fluctuation without tenderness at site of first puncture.

May 7.—A puncture was made into fluctuating space between 10th and 11th ribs at back, but only 1½ ounce of clear hydatid fluid, containing hooklets and echinococci, could be obtained.

June 8.—Patient left hospital of his own accord, feeling a good deal better, and with much less uneasiness in his side, but still jaundiced, and suffering from diarrhœa and flatulence.

In the three following cases there were numerous cysts in the liver, and in the peritoneum. From their size and other characters, those in the peritoneum appeared to be secondary in point of age to those in the liver. In Case XXVII. one large cyst in the liver was punctured with a fine trocar, and a free opening was made into a second cyst which suppurated.

Case XXVII.—*Multiple Hydatid Tumours of Liver and Peritoneum—Paracentesis of one Cyst—Suppuration of a second Cyst—Free opening —Death.*

Mary H——, 21, adm. into St. Thomas's Hosp. Sept. 25, 1873. Both father and mother had died of consumption. Four years ago noticed a swelling in right hypochondrium, which has continued to increase. A year later observed a second tumour in right inguinal region, and a third to right of umbilicus. For three years there has been swelling of veins of right leg, and for one year she has suffered from dyspnœa on exertion. Quite recently she has complained of sharp pain in right hypochondrium, and she has been losing flesh.

On admission, very weak; much pain in right side. Abdomen greatly enlarged; girth at umbilicus 40 in., and 2 in. above this 38½ in.; from ensiform cartilage to umbilicus 8 in., and from umbilicus to pubes 6½ in. In right hypochondrium and epigastrium is a large swelling, smooth, tense, fluctuating, and not tender. A second tumour, about size of an orange and quite movable, can be felt in right inguinal region; a third, somewhat smaller, to right of umbilicus, and two others in left iliac region. All these tumours are smooth, rounded, elastic, and distinct from one another. No ascites. Veins in walls of abdomen and chest enlarged. In front of right chest there is dulness on percussion, continuous below with that of aforesaid tumour, and ascending to second intercostal space. Posteriorly lungs resonant. Considerable dyspnœa. Apex of heart beats in fifth intercostal space, 2½ in. outside and on level with nipple. Appetite good; bowels regular.

Oct. 20.—Three pints of fluid drawn off by aspirator introduced at most prominent part of large swelling, 3 in. below ensiform cartilage and ½ in. to right of middle line. Fluid clear, alkaline; sp. gr. 1010; no albumen; much chlorides; no hooklets. After operation, girth at umbilicus 39 in., and two inches higher 37½ in.; from ensiform cartilage to umbilicus 7 in.

Oct. 22.—Much less pain, and breathing easier than before operation. Appetite good. Yesterday afternoon temperature rose to 100·6° and to-day it is 101·2°.

Nov. 13.—During last three weeks patient has had much fever, temperature varying from 99° to 104°; has often felt chilly, but has had no rigors or sweating. On Oct. 24 vomited once and had slight jaundice, which after a few days disappeared. On Nov. 3 patient still felt better than before operation; girth two inches above umbilicus 38¾ inches; but after this pain returned in large swelling, which grew rapidly until to-day, when girth 44 in., some œdema of legs, but no albuminuria. Aspirator introduced at same spot as before, and two pints of thin opaque yellowish fluid drawn off. No more could be obtained, as cannula became blocked. A free opening was now made into cyst by Mr. MacCormac, and nine pints more of fluid let out, but cyst was not emptied. A large perforated drainage-tube was fixed in opening. There was now clear percussion for 4 in. below right clavicle.

Patient did not rally; temp. did not rise above 100°, but pulse kept at 140 and was small and weak, until death on Nov. 15, 48 hours after operation.

Autopsy.—Peritoneum contained no fluid and was nowhere inflamed. Liver much displaced downwards and to left, and in great measure concealed by a large cyst, attached to its upper surface, which also encroached extensively on thorax. The external puncture had penetrated this cyst, the walls of which were collapsed, thin, and fibrous. It contained one large thick gelatinous hydatid, but no secondary cysts. It showed no sign of inflammation except a few small flakes of lymph adherent to its inner surface (outside hydatid). Behind this cyst was another, *almost as large*, and with much thicker walls, which had also been penetrated by the puncture. This cyst was also firmly attached to liver, and was in contact with posterior wall of abdomen; it contained one large hydatid, but no secondary cysts; its inner surface was intensely inflamed, partly villous, and plastered with large flakes of yellow lymph. Attached to under surface of left lobe of liver was a third cyst, size of a tennis ball, with thick walls, and full of cheesy matter and dried-up hydatid cysts. No cysts in interior of liver. Attached to peritoneum numerous cysts. Just below liver in front of right kidney were two—one as large as a cocoanut and containing clear fluid and numerous secondary cysts, and another somewhat smaller. Six or seven, of size of hen's egg or smaller, were attached to great omentum, and two grew from fundus of uterus and broad ligament, of which one contained cheesy matter and shrivelled cysts. Lower part of both lungs, especially right, collapsed. Other organs healthy.

In Case XXVIII. the history seemed to leave little doubt that the disease commenced in the liver, and that it was not until after many years that the peritoneum was secondarily invaded. The large cyst was punctured merely with the object of diminishing the abdominal distension and affording relief. But the cyst was already before death much smaller than it had been before the operation, and the *post-mortem* examination showed that its size was mainly kept up by secondary cysts in its interior.

Case XXVIII.—*Multiple Hydatid Tumours of Liver and Peritoneum—Ascites—Puncture of two Cysts and Paracentesis Abdominis.*

Charles M——, æt. 45, a teacher of languages, adm. into Middlesex Hosp. under my care, March 30, 1871. He had been a gymnast and a man of great muscular strength, and had never ailed in any way until 1857, when he noticed one morning while washing himself a tumour over left lobe of liver. This tumour was about size of half an orange, and quite painless; in fact, he would ont have been aware of its presence had it not been visible. It did not seem to grow, and in 1859 he first felt a little out of health, complained of pain under left scapula, of occasional dyspnœa, and of a stitch in region of tumour. In 1860 these symptoms got better, and he continued well until 1863, when, one morning, while rubbing his back with a towel, he experienced a dull pain in epigastric region, which increased and lasted for three weeks, and continued to recur occasionally for three years. During 1867 he suffered a good deal of pain in region of bladder, with frequency of micturition, and urine was high-coloured and deposited a reddish sediment. Early in 1869 he had first noticed a tumour similar to first in umbilical region, but this had never been seat of any pain.

For two years he had been very susceptible of cold, and in Jan. 1870 he had suffered from loss of appetite, cough, and from a severe pain in left side of chest, increased on inspiration. For twenty-five years patient had lived in Russia and in different parts of Europe, and had eaten food of countries in which he travelled.

On admission into hospital patient was of rather spare habit, but of unusually good muscular development. His sole complaint was of large size of abdomen, which presented a distinctly nodulated or botryoidal appearance. In first place there was a large prominence occupying space between sternum and umbilicus, but more to left than to right of middle line. This appeared to project from left lobe of liver, and to be as large as a child's head. It was fixed, and presented distinct fluctuation, but no vibration. A smaller rounded mass, about size of a cricket-ball, projected immediately below umbilicus; this was freely movable, and could be pushed to right or to left of middle line. A third, larger than an orange, could be felt projecting from margin of right lobe of liver, and separated from first by a distinct depression. A fourth, which seemed as large as a man's fist, was in left iliac region, but more deeply seated than the others. Two and a half inches below and to right of the umbilicus, was a fifth, about size of a hen's egg, freely movable and not causing any prominence on surface of abdomen. A sixth, movable and about size of half a walnut, appeared to be in abdominal parietes, over most prominent part of first, immediately to left of middle line (fig. 14). Other tumours of a similar nature could be obscurely felt in different parts of abdomen. All of tumours were rounded, soft, and elastic, and painless even on free manipulation. There was *no ascites,* and no jaundice. Girth of abdomen over most

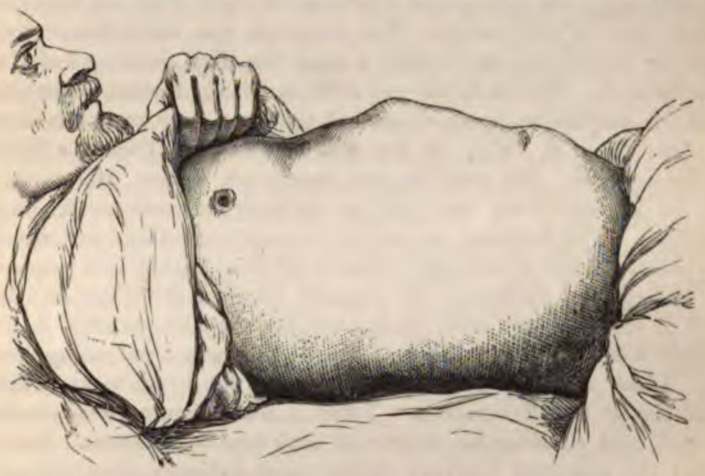

Fig. 14. The pointed prominence corresponds to the sixth tumour. From a photograph.

prominent part of first tumour 2 in. below sternum was 36½ in., and 1 in. below umbilicus it was 36 in. Measurement from lower end of sternum to umbilicus 6½ in.; from umbilicus to pubes 7½ in. The chief inconvenience which patient experienced from state of his abdomen was that of weight. Hepatic dulness did not ascend too high into chest, either in front or at back. Tongue clean; appetite good; occasionally slight acidity, but no other symptoms of indigestion; bowels regular; pulse 60; heart and lung signs healthy, except that breath-sounds were feeble at bases of lungs; urine free from albumen.

On April 3 a small trocar was introduced into small superficial cyst (sixth in above enumeration), and there escaped one and a half drachm of clear alkaline fluid, containing abundance of chlorides, but no albumen. On microscopic examination there were no hooklets or other signs of echinococci. The puncture was not followed by any pain or tenderness, and within a week the little swelling had almost disappeared. Patient would not consent to any further interference, and left hospital on April 10 in much the same state as on admission.

On March 6, 1872, he was admitted into St. Thomas's Hospital under my care. His condition was now much worse. His girth was 40 in. at umbilicus, and 40½ in. halfway between umbilicus and sternum. This increase was partly due to slight ascites, but mainly to an increased size of tumours; that in left iliac region now seemed to be as large as a cocoanut; the distance between umbilicus and lower end of sternum was now 8½ in., and the hepatic dulness in right nipple line now rose to level of nipple, and measured altogether 9½ in. Liver at some places felt extremely hard, while at others it was elastic and fluctuating. Veins in abdominal parietes much enlarged, and considerable œdema of both legs below knees. No jaundice; the abdomen nowhere tender; and patient's chief complaints were of occasional tightness of abdomen, and of pain in right shoulder. Pulse 84. Urine contained a small quantity of bile-pigment, but no albumen.

Abdomen continued to increase in size, so that on March 27 it measured 43 inches at umbilicus, and breathing was beginning to be embarrassed. A puncture was made on this day with a fine trocar into large cyst between umbilicus and sternum, and 28 fluid ounces of hydatid liquid were drawn off, having an alkaline reaction and a specific gravity of 1009, and containing much chlorides, but no albumen; no traces of echinococci on microscopic examination; but it was noted that there were constant interruptions to escape of the fluid by cannula. Girth at umbilicus immediately after tapping was reduced to 40½ in.

On March 29 temperature rose to 103·4°, and for a week it ranged between 101° and 102°, but no retching nor abdominal tenderness. The ascites, however, and enlargement of abdominal veins rapidly increased, and there was also a considerable increase of œdema of legs.

On April 1 girth at umbilicus 44 in., and on the 10th, 46 in. Orthopnœa set in; patient was obliged to sleep sitting up in a chair; and he occasionally suffered from severe attacks of suffocative dyspnœa. Under these circumstances paracentesis abdominis was resorted to, and 140 fluid ounces of serum were drawn off from peritoneum; alkaline, specific gravity 1016, and loaded with albumen. The operation gave great relief, and, what was remarkable, fluid did not reaccumulate in peritoneum. On morning after paracentesis girth at umbilicus was 41 inches, and on April 29 it was only 37½ inches. On latter date, also, girth round most prominent part of tumour in epigastrium was only 38 in., being 2½ in. less than at time he was admitted into hospital. The distance also from umbilicus to sternum measured only 7½ in., being 1 in. less than on admission. Œdema almost disappeared from legs, and patient was able to get up and walk about a little. Both before and after paracentesis patient took diuretics, including blue pill, squill, and digitalis.

On May 1 he seemed to be as well as usual, and got up for a short time in evening, but after getting into bed, his breathing became suddenly embarrassed, and in twenty minutes he was dead. His consciousness continued to the last, and he complained of no acute pain.

Autopsy.—Peritoneum contained less than a pint of clear straw-coloured serum. Nowhere any traces of recent peritonitis. Two enormous cysts in liver, one in front growing down from under surface of left lobe, and containing an enormous number of hydatid cysts, with a small quantity of thin pus, the entire contents measuring six pints. This was the cyst which had been tapped. The other cyst was in back part of right lobe, and contained between four and five pints of thin opaque fluid, in which there was bile-pigment, with a few hydatid cysts. Numerous smaller cysts were found in liver, and growing from omentum and other parts of peritoneum. Altogether there must have been many hundreds of them. There was one as large as a man's fist in spleen, and another still larger in left iliac region; another of size of a large orange, and quite globular, was attached by a narrow pedicle just below umbilicus; and two, as large as oranges, and with thick opaque white coats, lay quite loose in peritoneal cavity in right flank.[1]

Heart small and flabby; it contained no hydatids, and there was no thrombosis of the pulmonary artery. Lower part of both lungs condensed, apparently from pressure; and left lung everywhere firmly adherent to wall of chest.

Nothing was found to account for patient's sudden death.

Case XXIX. is remarkable for the successful excision by Mr. Spencer Wells of an immense number of hydatid cysts from the peritoneum.

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[1] See also Case XLIV.

CASE XXIX.—*Multiple Hydatid Tumours of the Liver (?) and Peritoneum, in part successfully removed by operation.*

Elizabeth C——, æt. 29, adm. into Middlesex Hosp. under my care on December 12, 1870. She had previously been in Samaritan Hospital, under care of Mr. Spencer Wells, who was good enough to transfer her to me.

On admission patient was pale, weak, and thin, but her countenance was not expressive of pain or cachexia. Her sole complaint was of swelling of abdomen, which measured 33½ inches at umbilicus. Abdomen generally was soft and elastic, and nothing like a solid tumour could be felt; neither was swelling due to an accumulation of gas in bowel, for greater part of it was dull on percussion; nor was it due to ascites, for as patient lay on her back there was tympanitic percussion in left flank. On careful inspection of abdomen swelling was seen not to be uniform, but was marked by a number of small rounded prominences, corresponding to rounded movable tumours, from size of a cherry to that of an orange, which could be felt in large numbers, and some of which were even visible through abdominal parietes (fig. 15.)

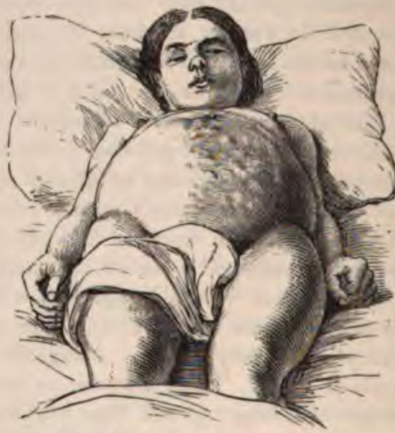

Fig. 15. From a photograph.

Several of these could be felt projecting from below right ribs, but whether they were attached to liver or not could not be determined. All of them were rounded and very elastic, and in one of largest, situated to right of umbilicus, distinct vibration could be made out on percussion. Hepatic dulness reached to upper border of fourth rib, but lower margin of liver could not be defined from dulness due to the nodular mass filling abdomen. Abdomen was nowhere tender on pressure, and only pain patient complained of was that of abdominal distension, increased after a full meal. Appetite fair; no evidence of disease of kidneys, or of thoracic organs; but patient suffered a good deal from dyspnœa, owing to pressure upwards against diaphragm, and apex of heart could be felt beating as high as third intercostal space.

The history which the patient gave was this:—Her father, mother, and three sisters were alive and in good health, and there was no history of phthisis or of cancer in family. Excepting diseases of childhood, patient herself had enjoyed good health until age of nineteen. She was married at eighteen; twelve months afterwards she gave birth to a child, and three months after this she awoke one night with severe pain below right ribs, vomiting, and faintness, and she discovered for first time a swelling of size of a hen's egg in situation of pain. After several days pain and vomiting subsided, but from that time patient had never been so well as she had been before; she had no definite illness, but felt weak and languid. The swelling below right ribs continued, without increasing notably in size, and three or four years afterwards she noticed a similar swelling in left iliac region, painless from the first, but which gradually increased in size. After this she had at varying intervals paroxysms of abdominal pain, not limited to situation of the appreciable swellings, but general. The pain would last for several days, and while it lasted she would scream out and retch very much. Nine months before admission she first noticed the numerous lumps scattered over abdomen, and about same time she began to lose flesh. After commencement of her disease patient became four times pregnant, and of her five children four were alive and in good health, the fifth having died of convulsions at age of a month. In intervals of her pregnancies catamenia had been regular, but for two years before admission, uterus had prolapsed when she walked about.

The diagnosis arrived at was that abdominal swelling was due to multiple hydatid tumours of liver and peritoneum, and that liver had in all probability been primarily affected. It was proposed to test this diagnosis by tapping one of largest tumours with a fine trocar, but the patient would not consent to this, and left hospital on Dec. 19.

On Dec. 31 she was re-admitted into Samaritan Hospital under care of Mr. Spencer Wells, who, on Jan. 12, 1871, tapped large cyst to right of umbilicus, and obtained one fluid ounce of clear hydatid fluid containing much chlorides, but no albumen. Finding no cyst large enough to make any difference in size of abdomen by tapping, Mr. Wells proceeded to make a small incision through abdominal parietes, and through this he removed three or four pounds of hydatid cysts[1] from omentum and mesentery, leaving at least as many more scattered all over abdominal wall, omentum, mesentery, and coats of intestines. The condition of liver and spleen was not observed during operation. Patient had no bad symptom; within ten days wound healed, and in less than a month she left hospital considerably relieved of the uncomfortable feeling of abdominal distension.

For nine months patient continued much relieved, but gradually abdomen again became enlarged, and about middle of October Mr. Wade, of Greenwich, who was called to see her, found her very prostrate, with great enlargement of abdomen, which had a distinctly nodulated appearance. After this she had persistent vomiting and purging, and she died at last by asthenia. There was no *post-mortem* examination.

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[1] *Now in* museum of St. Thomas's Hospital.

In Case XXX. a hydatid of the liver was evacuated by a large and permanent opening. The patient ultimately recovered, but from the history it seemed clear that subsequently to the operation a cyst in the liver discharged itself through the right lung. It is impossible to say whether this was the cyst which had been punctured. The fact that bile was expectorated, and that none was ever observed in the discharge from the external opening, suggests that it was not.

CASE XXX.—*Hydatid Tumour of Liver—Evacuation by a large opening—Subsequent bursting of Hydatid through diaphragm into Lung.*

Elizabeth R——, 39, lady's-maid, adm. into St. Thomas's Hosp. July 25, 1874. Father died at 71, and mother at 75. Two sisters and one brother died of consumption; two brothers and one sister alive and well. Although not very strong, patient had never been ill from childhood till two months ago, when she began to feel a sharp pain and a sensation of dragging down from shoulders in epigastrium and right hypochondrium, accompanied by occasional vomiting, and after a few days by slight jaundice. The jaundice and sickness soon subsided, but the pain persisted. A few days after commencement of pain, she first noticed a swelling below right ribs—considerable at first, but which gradually increased up to admission.

On admission, still complains of pain as described above. Projecting downwards from right lobe of liver is a tumour, about size of a cocoanut, globular, smooth, very elastic, and not tender. No jaundice or ascites. Pulse 108. Temp. 100·4°.

During August and September I was absent from hospital, and but few notes of patient's case were recorded. On Aug. 15 tumour was noted as tender; and on Aug. 24, patient having again become slightly jaundiced, tumour was punctured, first with a small and then with a large trocar, and the contents evacuated. These consisted of a clear fluid with a number of hydatid cysts and echinococci. A cannula was tied in, the cavity was washed out from time to time, and an ice-bag applied over part.

On Sept. 5 it was noted that patient was very weak and anæmic, and pulse feeble. Since Aug. 28 there had been no jaundice. On Sept. 14 she again complained of great pain in right side, with fever (temp. 101°). She had also frequent cough and expectorated mucus of a bright yellow colour from admixture of bile. Over lower half of right lung at back there was dulness on percussion with feeble breathing, diminished vocal fremitus, and distant crepitation. Anteriorly also there was dulness as high as nipple. The pain and fever subsided after about a week, but on Oct. 6 she was still expectorating bile, and the dulness and other signs remained at base of right lung. The external opening was now closed, and at no time had there been any discharge of bile by it. No hydatid membrane or signs of echinococci had been found in sputum, but it contained bile up to Oct. 12. Patient continued for a long time very weak, and was not able to leave her bed until Nov. 21, by which time base of right lung had become clear. On Dec. 18 the catamenia returned after an absence of six months, and by the end of the month she was able to leave the hospital.

In the four following cases a hydatid of the liver opened into the bile-duct, which became obstructed by hydatid vesicles, so that jaundice resulted. In Case XXXI. the liver contained three cysts, two of which communicated with one another, and had suppurated before the patient came under observation. Paracentesis was resorted to solely as a palliative. The case was also interesting from the circumstance that the patient's brother had also a hydatid of the liver.

CASE XXXI.—*Suppurating Hydatid Tumours of Liver, one opening into Lung and another into Bile-duct—Jaundice—Temporary relief from Paracentesis.*

Charles W——, aged 24, adm. into Middlesex Hosp. May 19, 1869. Had enjoyed good health until 12 months before, when, after getting wet in a thunderstorm, he was laid up for two months with inflammation in chest, on recovering from which he suffered for a week from headache and vomiting, and ever since he had complained of general debility and lassitude. Eight months before admission he began to complain of vomiting in morning, and drowsiness and tightness at epigastrium after meals, but notwithstanding persistence of these symptoms he continued at work until three weeks before coming to hospital, when symptoms became worse, he lost flesh and suffered from great itchiness of skin, and first became aware of a swelling in right hypochondrium, which had continued to enlarge up to admission. Ten days before admission, jaundice appeared, and his motions became slate-coloured.

On admission, patient was deeply jaundiced, prostrated, thin, and with anxious pinched features. Liver appeared enormously enlarged, so as to cause a visible bulging of upper part of abdomen. Following measurements were taken in recumbent posture. Girth at umbilicus 36 in.; half-way between umbilicus and sternum, 38¼ in.; at lower end of sternum, 37½ in.; from lower end of sternum to umbilicus, 7¼ in.; from umbilicus to pubes, 6 in. Hepatic dulness in r. m. l. commenced an inch above nipple and extended to half an inch below umbilicus, measuring 12¼ in. Lower margin of liver rounded; surface smooth and painless, and to right of middle line distinctly fluctuating and yielding 'hydatid vibration.' Projecting from lower margin of left lobe was a circumscribed fluctuating bulging about size of an orange, apparently distinct from cyst in right lobe. Also, behind large cyst on right side could be felt another rounded projection from liver, without any distinct fluctuation. Peritoneal hepatic dulness ascended as high as 5th dorsal spine. Subcutaneous veins in right axillary and lumbar regions enlarged, and evidence of slight ascites. Tongue clean; appetite fair, but afraid to eat much on account of painful feeling and distension; 3 or 4 loose motions daily, devoid of bile. Urine loaded with bile-pigment, but free from albumen. Pulse 80; heart elevated, its apex beating between 3rd and 4th ribs. Considerable dyspnœa, and slight crepitation at base of right lung.

May 27.—Diarrhœa has persisted, but dyspnœa and feeling of abdominal tightness have increased. An exploratory puncture was made to-day into cyst in right lobe of liver. Seven ounces of fluid were drawn off, viscid, yellowish, and containing pus-corpuscles, shreds of hydatid membrane, hooklets, and rhomboidal blood crystals.

May 29.—Pulse 84; temp. 98·2°. To-day a fine trocar was passed into cyst in left lobe. Only a few drops of bright yellow matter of the consistence of clotted cream could be squeezed out; this contained oil-globules, nuclei, and abundance of hooklets, and minute red rhombic crystals.

June 3.—Cyst in right lobe was again opened with a large trocar under ether spray. Eighty ounces of yellow pus were let out, containing numerous collapsed hydatids and fragments of cysts; last few ounces were deeply tinged with blood. In consequence of hæmorrhage cannula was withdrawn and wound closed with collodion, although cavity did not appear nearly emptied.

June 8.—Pulse 96; temp. 98·2°. Great relief followed last operation, but, cyst apparently filling again, it was determined to make a free opening into it. On again introducing large cannula, much blood, partly clotted, escaped with hydatid membranes, and after 4 ounces had been drawn off opening was again closed.

June 18.—Patient left hospital at his own request. Still deeply jaundiced and suffering from diarrhœa. Temperature since last operation has never exceeded normal standard. Girth over most prominent part of tumour, 38 in.

June 28.—After discharge, tumour continued to enlarge, and to-day patient's wife came to say that he had been suddenly seized with most profuse diarrhœa and had passed 'pieces of skin' and 'bits of jelly' in motions, while at same time swelling in side had become suddenly less.

July 3.—Much thinner and weaker, and has no appetite and much thirst, but jaundice almost gone. Girth over most prominent part of tumour now only 34½ inches. Right lobe of liver comparatively flat, so that cyst in left lobe appears much more prominent. Diarrhœa continues, but less profuse. The same evening patient became suddenly collapsed, and died on evening of *July* 4.

Autopsy.—Girth of abdomen over most prominent part of tumour, 33¾ in. Abdominal parietes and transverse colon firmly adherent to right lobe of liver, and also some recent lymph over rest of liver and adjacent bowels. Peritoneum contained a pint of fluid. Mucous membrane of adherent colon intact. Duodenum contained bile and several small hydatid cysts, and dark green bile could be squeezed into it through dilated orifice of bile-duct. Common bile-duct greatly dilated, so that a No. 8 bougie could be passed with ease through duodenal opening into large cyst in right lobe. This cyst was as large as a child's head and contained much reddish-brown pus, with numerous hydatid cysts up to size of a small orange, some collapsed, but others full and plump. The parent cyst was collapsed and ruptured, having separated from wall of cavity, which was lined with flocculent lymph. This large cavity communicated with another almost as large, also in right lobe of liver, but higher up, by a well-defined circular opening just large enough to admit tip of finger. This cyst had similar contents to first, and was firmly adherent to diaphragm, which in its turn adhered firmly to base of right lung. The diaphragm was at this part perforated, and in the opposed part of lung was a cavity, the size of an apricot, with ragged walls of pulmonary tissue and traversed by bands of disintegrating lung. There was no fluid or hydatids in right pleura. These two cysts occupied greater part of right lobe of liver. Left lobe of liver was also much enlarged, and projecting from its anterior margin was a third cyst, larger than a man's fist, with thick walls infiltrated with calcareous matter, and its interior full of a bright yellow pulp containing innumerable hooklets of echinococci and blood-crystals. Other organs healthy.

In June 1870, Thomas W., aged 27, brother of above patient, consulted me on account of an elastic fluctuating tumour in epigastrium, the size of a cocoanut, and causing a bulging of costal cavities on both sides. He had first noticed it four months before. It was unattended by pain or constitutional symptoms. He had lived for a short time with his brother 2½ years before, but never before then during 14 years. He would not consent to any operation.

In the following case, which you must all have watched with much interest, we were enabled to diagnose during the patient's life that a communication had been established between the tumour and the common bile-duct. The fact that the tumour had undergone suppuration and that the contents were fetid contra-indicated the ordinary operation, and compelled us to substitute a large permanent opening.

Case XXXII.—*Hydatid Tumour of Liver, opening into the common Bile-duct.—Jaundice and Suppuration of Cyst—Puncture with a large Trocar, and permanent Opening—Pneumonia—Death.*

On February 4, 1868, I was requested by Dr. Ayling, of Great Portland Street, to see Mrs. C——, aged 30, who was suffering from jaundice and enlargement of liver. Her mother stated, that ever since she had been fourteen there had been a fulness in epigastrium and left hypochondrium, but that, with exception of occasional pain after food and other symptoms of indigestion, she had enjoyed good health until present illness. She had been married for eleven years, and during that period catamenia had been regular, and she had had no children or miscarriages. Eighteen days before I saw her, she had been suddenly seized with severe pain in back and upper part of abdomen, which almost bent her double. This was relieved by warm poultices &c., but was soon followed by pyrexia, and four days later by jaundice, which soon became intense, with dark porter-coloured urine, and a complete absence of bile from motions. The fever continued; the swelling in epigastrium and left hypochondrium was observed to increase, and patient was so prostrate that some days before I saw her she was thought to be sinking; but she had no vomiting, rigors, or night-sweats.

I found patient much emaciated, and with deep jaundice of conjunctivæ and whole surface of body. There was a distinct tumour in epigastrium, extending apparently into both hypochondria. It projected forwards fully 1½ in. beyond natural level, and pushed forward lower end of sternum, and lower ribs on both sides, but particularly on left. When patient lay on her back, lower margin of tumour was 1 in. above umbilicus. Tumour was evidently connected with liver, dulness of which in mesial line was 9 in., in right mammary line 5 in. and in left 6 in. Posteriorly and laterally hepatic dulness did not rise higher than natural on right side, but on left posteriorly, it was fully 2 inches higher than on right, and the dulness in left axillary line was 9 in. The tumour, where it presented itself at epigastrium, was rounded, smooth, and slightly tender. Distinct fluctuation could be felt in it, and a thrill, as from fluid, could be made out in epigastrium when percussion was made over the dull part at back of left side of chest. Tongue very red and clean, with enlarged papillæ at tip; centre smooth and deeply fissured. Motions clay-coloured, without a trace of bile-pigment. Pulse 108. Apex of heart elevated by tumour to between fourth and fifth ribs. Respirations 28, and slightly embarrassed, but pulmonary signs normal. Temperature 100·6°. Urine 1027, containing both bile-pigment and bile-acids (Harley's test), but no albumen.

The fact that tumour contained fluid, and had probably existed for years without giving rise to symptoms, indicated hydatid; the acute pain, followed by jaundice, with disappearance of bile from stools, made it probable that this hydatid had communicated with and obstructed the main bile-duct; while the enlargement of tumour, with fever and great prostration, was accounted for by inflammation of tumour, consequent on entrance of bile. This was the diagnosis.

On following day patient was admitted, under my care, into Middlesex Hospital, and as her condition became daily more critical, it was determined to have recourse to puncture of the tumour, as holding out the only chance of safety. Accordingly, on Feb. 7, a fine trocar was introduced by Hulke in left side of epigastrium, and about six ounces of fluid drawn off. This was deeply tinged with bile and very fetid, and contained numerous pus-corpuscles and scales of cholesterin, but no hooklets or echinococci. On ascertaining nature of fluid small cannula was withdrawn, and a full-sized trocar substituted. Several hydatid vesicles escaped through the larger tube, but only about eight ounces more of fluid, although a probe could be passed in 6 or 8 in. It appeared, therefore, that contents of cyst consisted mainly of hydatid vesicles. A solution of carbolic acid (2 per cent.) was injected into cavity, and a large tube was tied into wound.

During the ten days that followed operation, several pints of the carbolic acid solution were injected three times a day through an elastic catheter passed into cavity, and on each occasion large numbers of hydatid vesicles (with hooklets and echinococci in some) came away, with a fetid, purulent fluid, containing a large quantity of green bile. While this was going on, abdomen returned to almost its normal dimensions, and the jaundice in a great measure disappeared from integuments and urine, but motions remained as light as before.

Patient had repeated doses of morphia after operation, and for four days pulse was about 108, temperature was normal, and no very bad symptom, except development on tongue and inside of mouth of numerous aphthous ulcers on a raised base, which caused excruciating pain whenever she took food or drinks; but both pain and ulcers almost disappeared after repeatedly washing out mouth with Condy's 'ozonised water.' During night of Feb. 11, patient suffered from repeated rigors, and after this pulse rose to 140, respirations became quick, and tongue dry; occasional vomiting, and prostration rapidly increased. On morning of 18th delirium set in, and at 6 P.M. she died.

On opening abdomen, peritoneum contained no fluid, and there was no sign of recent peritonitis, but there were firm adhesions between tumour and diaphragm and abdominal parietes in front. Left lobe of liver had disappeared, and its place was occupied by an enormous hydatid cyst. This cyst contained about two pints of very fetid thick green fluid, with large fragments of parent hydatid cyst lying loose in cavity. It opened externally through wound in abdominal wall, while internally it communicated with common bile-duct by an opening large enough to admit a full-sized catheter. On slitting open duodenum, orifice of duct was found sufficiently dilated to admit a goose-quill, but obstructed by a large hydatid cyst, partially protruded into duodenum.[1] Between this and the opening into cyst, duct was distended with hydatid vesicles. Bile-ducts throughout liver greatly dilated, and liver itself very fatty and intensely jaundiced, with a tight-lace prolongation downwards of right lobe. No trace of bile-pigment in intestinal contents. Spleen adherent to tumour, but otherwise normal; kidneys healthy. Recent pneumonia, at some places passing into condition of grey hepatisation, of back of lower lobe of both lungs and of upper lobe of right.

The following case was in some respects very similar to the last. The attack of diarrhœa was probably due to the partial discharge of the contents of the cyst through the bile-duct into the bowel.

CASE XXXIII.—*Hydatid Tumour of Liver opening into Bile-duct—Jaundice from obstruction of duct by Hydatid Membrane—Pyæmia.*

Jane R., 33, charwoman, admitted into St. Thomas's Hosp. Nov. 13, 1874. Nothing of importance in family history. Married and had five children, of whom three are alive and well. Had 'typhoid fever' two years ago, but on whole had good health till eight months ago, when she began to complain of an occasional gnawing pain in right hypochondrium, which after three months spread up to the shoulders. These pains did not prevent her going about following her work, but she was usually ailing and she lost flesh. A month before admission the pain became much increased, and after a fortnight she was suddenly seized with very acute shooting pain in right hypochondrium attended by rigors and vomiting, and followed after two days by jaundice, urine like porter, and white stools. She had kept her bed from commencement of this acute attack until admission. Six weeks before admission, she first noticed a tumour in right lumbar region, which was then comparatively small, and appeared to be movable. This rapidly increased in size, especially during last three weeks.

State on Admission.—Very prostrate and emaciated, but did not complain of much pain. Decided jaundice of skin and conjunctivæ. No dropsy. Tongue clean, but dry down centre; sordes on lips; no appetite; bowels confined; an enema brought away some white fæcal matter. Hepatic dulness much increased, commencing ½ inch below nipple and extending 9½ in. downwards, or to 2 in. below level of umbilicus. Portion of liver below ribs causes a distinct bulging of abdominal parietes; its surface firm, smooth, and somewhat tender. Left lobe of liver not enlarged, the tumour turning abruptly upwards at umbilicus towards ensiform cartilage. No induration of integuments around umbilicus; no ascites; no enlargement of abdominal veins; splenic dulness not increased; no sign of any bowel in front of tumour and distinct tympanitic percussion behind it. Thoracic organs healthy; resp. 20; pulse ranges from 92 to 128, and temp. from 97° to 100°. Urine retained, or passed involuntarily, 1017, contains much bile-pigment and lithates and ⅛ albumen, and also a few blood-corpuscles and epithelial scales, but no leucin or tyrosin. Occasionally delirious, and mind at all times so confused that it is impossible to obtain a clear account of her illness.

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[1] The preparation is in the museum of Middlesex Hospital. In the museum of St. Bartholomew's Hospital is a specimen (xix, 12) of hydatid tumour of right lobe of liver opening into bile-duct, which is blocked up by hydatids, one of which projects from the orifice of the duct into duodenum, as above.

On Nov. 17 patient had rather a severe attack of diarrhœa, which lasted for about three days, during which she passed in bed numerous black, liquid, very offensive motions, which unfortunately were not carefully examined. On Nov. 20 diarrhœa had ceased, and motions were again noted as light and solid. On Nov. 21 patient appeared slightly better and her mind was clearer; but after this her prostration increased and she gradually passed into a state of stupor, with dry tongue and sordes about mouth, which continued until death on Nov. 25.

Autopsy.—Right lobe of liver greatly elongated, extending 9 in. below level of lower end of sternum. Projecting from its anterior margin, and looking very much like gall-bladder, was a hydatid cyst, about 2 in. in diameter. Another cyst, larger than a man's fist, was embedded in substance of right lobe, projecting slightly from upper and anterior surface. This cyst contained a thin purulent-looking fluid coloured with bile and several smaller cysts, and the cavity in which it was lodged communicated with the bile-ducts. A large mass of hydatid membrane blocked up the termination of common duct, and partially projected into duodenum. The common, cystic, and hepatic ducts were all greatly dilated, and in interior of liver ducts were also dilated into cyst-like cavities filled with opaque orange-coloured fluid. Gall-bladder contained three calculi and several small hydatid cysts. Kidneys congested and stained with bile, but otherwise healthy. Stomach, spleen, heart, and brain normal. Lower lobe of right lung adherent to diaphragm, much congested, and containing several solid blocks, one hæmorrhagic, and the others dark red with a drop or two of dirty pus in interior. Lower lobe of left lung congested, but free from blocks.

The following case, XXXIV., is remarkable no less for the fact that the patient recovered after discharging the contents of a large hydatid of the liver through the bile-duct into the bowel, than for the extraordinary manner in which death ultimately occurred.

CASE XXXIV.—*Hydatid Tumour of Liver, bursting into Bile-duct—Jaundice—Discharge of numerous Hydatid Membranes per Anum—Recovery—Attacks of Biliary Colic from passage of Cysts remaining in Liver through Bile-duct—Rupture of old Adhesions of Liver during act of Vomiting—Peritonitis—Death.*

On October 29, 1861, I was consulted by Mr. G. W——, a solicitor, aged 53. For some weeks he had been suffering from flatulence and a feeling of tightness and oppression after meals, and three days before he had been attacked with severe pains in abdomen, resembling colic. The countenance was somewhat sallow; motions were pale, but contained bile; no bile in urine, which was scanty and dark, having a specific gravity of 1027, and depositing much lithic acid. Vertical hepatic dulness in the right mammary line extended about an inch below edge of ribs, and all along right hypochondrium there was slight tenderness on pressure. Pulse 64. His digestion had always been good, except once, about seven years before, when he had several attacks of colicky pain in abdomen, similar to those from which he had recently suffered. The remedies prescribed by myself, and afterwards by Sir Thomas Watson, who met me in consultation, failed to give relief.

On Nov. 24 patient had an attack of vomiting, followed by an aggravation of the dyspeptic symptoms, and by increased tenderness in right hypochondrium.

On Dec. 6 he was much worse. The tenderness in right side had increased greatly, and there was also constant pain there, which became very acute when he took a long breath or coughed. Tongue furred and moist; bowels very costive; considerable tympanitic distension of abdomen, and increased sallowness, but no sickness. Pulse 88; respirations 30, and thoracic. Fifteen leeches were applied to seat of pain; twelve more on Dec. 8, and eight more on Dec. 10, with poultices in the intervals, and bowels were kept open by castor-oil and turpentine enemata.

On Dec. 12 pain was much less, but there was still considerable tenderness and a stitch in right side on taking a breath or coughing. Countenance very sallow, but no decided yellowness of conjunctivæ, and motions, though pale, contained bile. Vertical hepatic dulness in right mammary line 5 in. Nothing like a defined tumour could be felt, and there was no bulging of ribs. Breathing at base of right lung normal. Pulse 88.

On Dec. 16 and 17 patient passed, for first time, several hydatid cysts in a bilious motion.

On Dec. 18 he was much worse. There was decided jaundice of integuments; urine loaded with bile-pigment, and not a trace of bile or of hydatid membranes in motions. Constant pain in right side, in addition to occasional paroxysms, like colic; lips parched; tongue furred; much perspiration in night, and great prostration. Pulse 100.

Treatment consisted in constant application of poultices to side, and in administration of blue pill and opium.

Dec. 19.—Is much easier. Has passed a large quantity of hydatid vesicles, from a pin's head to an orange in size, per anum. Skin and urine still jaundiced, and no bile in stools.

20.—Fæces to-day are tinged with bile, and still contain numerous hydatid cysts.

21.—Jaundice almost gone. Motions still contain hydatids and abundance of bile. Below and to left of right nipple, tympanitic percussion over a space the size of a crown-piece. Both above and below this there is hepatic dulness. Pulse 88; pain much less; tongue cleaning.

The patient continued to pass a few hydatid vesicles with each motion up to Dec. 31, and the tympanitic percussion sound above noted remained a few days later than this. He had occasional sharp, but temporary, attacks of pain in abdomen, resembling colic. On Jan. 6, 1862, he was quite convalescent. Pulse 72. The tympanitic sound noted above could no longer be distinguished, and upper border of hepatic dulness was an inch lower than before. At end of Jan. Mr. W—— was able to drive out; and on Feb. 19 he went to Ventnor for change of air, returning to London on March 11.

Once, while at Ventnor, he had a severe attack of colicky pain lasting for an hour and a half, and 'bending him up double.' He had a similar attack, but less severe, a few days after his return to London. Both attacks were unaccompanied by vomiting. Every day he gained strength, and on his return to town he was able to resume his business. On April 2 he went down to Essex on business. He walked about the country several miles every day, feeling none the worse, and returned to town on April 6.

On April 8 he went to his business as usual, and walked several miles. Shortly after dinner, about 7 P.M., he was suddenly seized with severe pain in abdomen, which returned in paroxysms, and this time was accompanied by vomiting. There was slight tenderness at epigastrium, but no jaundice. Pulse only 84. Repeated doses of opium and chloric ether were prescribed, and poultices were kept constantly applied over abdomen.

On following day, the paroxysms of pain had ceased, but there was more tenderness at epigastrium and in right hypochondrium, and considerable pain when he coughed or moved. The vomiting had not quite ceased. There was slight sallowness, but stools contained bile. Pulse 86. Ten leeches were ordered to be applied to side, and the poultices and opiates were to be continued.

The patient did not apply the leeches, as he felt better. In the afternoon, he had two severe attacks of rigors, after which he felt so much better and free from pain that he thought it unnecessary to send for *me*.

On the morning of April 10 he said that he felt so much better that he had eaten a good breakfast, and he wished to get up and go down stairs; but he was in a state of extreme prostration, and evidently sinking. The pulse was 120 at the elbows, and imperceptible at the wrists. The sickness had ceased, but the features were pinched, and the skin was cold and covered with clammy sweat. He gradually sank, and died at 8 P.M.

Autopsy.—Abdomen only was examined. On opening this cavity intestines appeared healthy, but distended with gas. No exudation or increased vascularity in general cavity of peritoneum. Large intestines contained a quantity of pulpy material of colour of cream, and without any tinge of bile. Small intestines contained bile.

Left lobe of liver was healthy and non-adherent. Both the upper and under surfaces of right lobe were firmly adherent to adjoining parts. Near right edge of liver a few of the bands of adhesion fastening it to ribs appeared to be ruptured, and at this point there was a patch of recent lymph not larger than a square inch, with slightly increased vascularity round about. In substance of right lobe was an irregularly-shaped, collapsed cavity, the size of a large orange; walls of this cavity were partly formed by ribs and surrounding adhesions; its inner surface consisted of indurated hepatic tissue, presenting a shreddy appearance, and not lined by hydatid membrane. The cavity was almost empty; but it contained four or five collapsed hydatid vesicles about size of a shilling. Communicating with it was a greatly dilated bile-duct, passing directly on to the common duct. The entire duct, from the cavity to the orifice in duodenum, was large enough to admit tip of little finger.[1] Further back, in right lobe, and quite distinct from cavity now described, was another, about size of a plum, which was lined by an obsolete and cribriform hydatid cyst, presenting a tough, opaque yellow appearance. The contents of this cavity had escaped during the hurried division of the liver. (This tumour may have been the source of the symptoms from which patient had suffered seven years before his death).

Case XXXV., like Case XXV., illustrates the good effects of antiseptic treatment after the free opening of a large hydatid which had undergone suppuration.

CASE XXXV.—*Suppurating Hydatid of Liver. Free Incision—Recovery.*

Miss M——, aged 24, consulted me on Nov. 24, 1869. She stated that for two years she had noticed a bulging of the lower right ribs. This had come on without pain, and had not increased much since it had been first observed. The bulging was quite obvious: the girth of the right side of the chest below the breast was 15½ in., of the left 13½ in. The hepatic dulness in the right nipple line extended from the nipple 8 in. downwards; it did not rise too high at the back, and its upper border was arched. The intercostal spaces over the bulging were obliterated, but nothing like fluctuation could be made out. There was no tenderness on pressure, and the general health was good.

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[1] The preparation is in museum of Middlesex Hospital.

I saw nothing more of the patient until Feb. 4, 1873, when she stated that for two months she had been liable to attacks of severe pain, shooting from the back to the front of the swelling, which made her scream and prevented sleep. These attacks were very apt to come on when she lay down. She had also uneasiness in the stomach after food, and was losing flesh. The swelling had increased especially in an upward direction. Below the breast the girth on the right side was 16 in. and on the left 14½ in. The hepatic dulness in front rose to 1½ in. above the nipple, and from this extended 9½ in. downwards, to 3 in. below the ribs; posteriorly also the hepatic dulness rose an inch or two above its normal level, and air entered imperfectly into lower lobe of right lung. No perceptible fluctuation. Pulse 108, temperature somewhat elevated. I expressed the opinion that the tumour was hydatid, and advised that an exploratory puncture should be made into it. Sir W. Jenner, who saw the patient with me on Feb. 22, coincided in this opinion and advice. On Feb. 24 the swelling was punctured between the sixth and seventh right ribs in front; 3 drachms of pus escaped; the opening was closed, and on Feb. 26 a free incision was made at the same spot by Mr. De Morgan, and 4 pints of pus containing numerous large hydatid cysts were evacuated. The cavity was washed out with a solution of chloride of zinc (20 gr. to ℥j), and a piece of elastic tube was left in the opening, through which a weak solution of carbolic acid was daily injected, and external opening was covered with carded oakum. The severe pain from which the patient had previously suffered was at once relieved. Much pus and hydatids continued to be discharged until *March* 17, when what appeared to be the parent sac escaped. After this the discharge rapidly diminished, and the patient began to gain flesh. On April 28 the tube was removed, and soon after opening closed.

On April 8, 1875, patient was in enjoyment of excellent health.

In Case XXXVI. the hydatid tumour not only suppurated, but induced pyæmia, with secondary deposits of pus throughout the liver.

CASE XXXVI.—*Suppurating Hydatid Tumour of Liver—Pyæmia, with secondary Deposits of Pus.*

Thomas B——, aged 35, was admitted into the London Fever Hosp. on Jan. 20, 1866. He had lived for twenty years in Tasmania, but for the *last four* years in England. His previous health had always been good. His illness commenced five weeks before admission with severe pain in right side, followed three weeks later by jaundice and diarrhœa. When seized with the pain, he first noticed a swelling in right side; but this was as large then as at time of admission. Patient was emaciated and jaundiced, and liver was much enlarged, vertical dulness in right mammary line being eight inches. The portion of liver projecting below right ribs was smooth, painless, elastic, and almost fluctuating, but yielded nothing like 'hydatid vibration.' Moderate ascites. Pulse 96; tongue moist and red; no appetite; six or seven liquid stools daily, containing little or no bile. Considerable sweating at night. Three or four days after admission, irregular attacks of rigors set in; diarrhœa continued; emaciation and perspirations increased; tongue became dry and brown; and on Feb. 22 patient died. On two occasions (Jan. 31 and Feb 7) an exploratory puncture was made into tumour. On first occasion nothing came away, owing to trocar being too short; on second occasion about six ounces of thin purulent bilious fluid were drawn off, which, unfortunately, was not submitted to microscopic examination. No bad consequence appeared to follow either operation.

At the autopsy, a hydatid cyst, as large as a child's head, and full of pus and secondary hydatids, was found projecting from under surface of liver, compressing portal vein and bile-ducts. The liver was studded with numerous small abscesses, and its outer surface was coated with recent lymph. Traces of the punctures were discovered with difficulty, and there was no evidence of increased inflammatory action in their neighbourhood.

In the following case the suppuration of a hydatid appears to have induced pyæmia, with secondary gangrenous abscesses in the liver. The anatomical characters of the liver agreed with those of 'gangrene of the liver' as described by Rokitansky.[1] This disease, however, is so rare that experienced observers have denied its occurrence, and Frerichs makes no mention of it. Even Rokitansky had met with only one example, and there it was associated with pulmonary gangrene. Budd reports one case, and quotes another from Andral.[2] Considering the rarity of such cases, the remarkably fetid odour observed during life is of clinical interest.

CASE XXXVII.—*Suppurating Hydatid—Pyæmia, with secondary Gangrenous Abscesses in Liver.*

A man, aged 27, was adm. into London Fever Hospital under my care, Feb. 23, 1867. He was so prostrate that he could give little account of himself, and all that could be ascertained was that he had been a soldier in the West Indies for about seven years, but that his health had been good until about a month before admission, when he was seized with pain in epigastrium and right hypochondrium, with nausea and vomiting, and about same time he first noticed a tumour below right ribs, pain in which made it difficult for him to button his tunic over it. On admission he lay on his back, with his legs drawn up; abdomen full and tender all over; friction heard distinctly over liver, which appeared large, extending downwards to crest of ilium, and upwards to lower border of third rib. Tongue dry and brown; frequent vomiting; but no jaundice, and bowels stated to be regular. Splenic dulness increased. Pulse 132 and feeble; heart's sounds normal; respirations quick and thoracic; dulness on percussion over back of right lung, and moist sounds heard over greater part of both lungs. Skin hot, face pale: features pinched.

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[1] Path. Anat. Syd. Soc. Trans. vol. ii. p. 136.

[2] Budd, op. cit. 3rd ed. p. 129.

On following morning prostration had increased, and, in addition, there was noted slight jaundice of conjunctivæ, and a peculiar, very fetid odour—*sui generis*, which appeared to proceed from entire body, and not from breath in particular. This was noted in case-book before patient's death, which took place on same day.

On post-mortem examination, which was made on day after death, considerable evidence of recent peritonitis, particularly in neighbourhood of the liver. Projecting from under surface of right lobe of liver, and but slightly embedded in it, was a hydatid cyst, larger than a cocoanut. The wall of the parasite was opaque, tough, and cribriform, from presence of numerous large openings, and its interior was filled with dirty brown purulent fluid, having a very offensive odour. Entire liver studded with numerous softened masses from the size of a nut up to that of a small orange, in which hepatic tissue was softened, and consisted of a spongy material, corresponding to the fibrous stroma and vessels, saturated with a greenish, extremely fetid, pulpy fluid. Embedded in substance of liver, near anterior edge of right lobe, was a healthy hydatid cyst, about the size of a chestnut, containing clear fluid and ecchinococci. Lungs congested, but nowhere inflamed or gangrenous.

In the following case the hydatid tumour was so large as to almost fill the abdominal cavity, and bile had entered the cyst. The real nature of the case was not recognised during the patient's life, and paracentesis was resorted to merely as a palliative to relieve the patient's extreme distress, and with no idea of effecting a cure.

Autopsy.—Heart normal. Left lung firmly and universally adherent: its lower lobe hyperæmic, and near base its tissue sank in water; but it was not granular on section, and it was unusually firm and tenacious. Right pleural cavity filled with pus, floating in which were innumerable hydatid vesicles, from size of a pin's head to that of an orange. Right lung completely collapsed and carnified, except at apex, which contained a little air. Liver much depressed, its lower margin reaching to more than half-way between umbilicus and pubes. Projecting from posterior margin of right lobe was a cyst as large as a child's head, and firmly connected to diaphragm; liver not adherent at any other part of its surface. At upper part of cyst there was a rupture through diaphragm, measuring one inch and a half in diameter, by which cyst communicated with right pleura. The interior of cyst was lined with a hydatid membrane; its cavity was filled with pus and vesicles. A large number of the vesicles were examined with microscope, but no echinococcus or hooklet could be discovered. No other hydatid tumour either in liver or in any organ of body. Pelvis and calices of right kidney and upper part of right ureter dilated, apparently owing to pressure below of the displaced liver; secreting tissue of right kidney much atrophied; left kidney normal.

CASE XL.—*Hydatid Tumour of Liver, opening into Right Pleura—Empyema—Pericarditis.*

George K——, aged 54, a gardener, of sober habits, adm. into Middlesex Hosp. under Dr. F. Hawkins, April 25, 1854. He had always enjoyed good health until four months before admission, when he was suddenly seized with pain all over abdomen, but particularly in right hypochondrium, and extending thence to right shoulder. About same time he became slightly jaundiced. The pain and jaundice continued; and at time patient came under observation he was very weak and emaciated, and suffered from incessant cough. Liver much enlarged, extending down to umbilicus. Considerable bulging of right side of chest, which was universally dull on percussion, and devoid of respiratory murmur, except at upper and back part close to spine. Patient gradually sank, and died on May 10.

Autopsy.—Right pleural cavity was filled with a yellowish, turbid, semi-purulent fluid containing masses of a gelatinous substance, which proved to be hydatid cysts. Right lung compressed and flattened against vertebral column, and at its base was firmly bound by adhesions to diaphragm. It did not crepitate in the least; it sank in water, and was completely carnified. Liver enormously enlarged, extending down as far as umbilicus, and weighing 90 ounces; it was firmly adherent to diaphragm. In posterior part of right lobe was a cavity as large as a swan's egg, lined with a hydatid cyst, and containing similar cysts in its interior. Upper wall of this cavity was formed by the diaphragm, and here there was a large opening by which the cavity in the liver communicated with right pleura. The liver was much congested. Pericardium glued to heart by recent soft adhesions. Left lung, spleen, and kidneys healthy.

CASE XLI.—*Old Hydatid (?) of Liver, communicating with Base of Right Lung—Lobular Pneumonia and Gangrene of Lung.*

Robert J——, aged 72, was sent to London Fever Hospital, August 21, 1864, as a case of 'fever.' On examination, he was found not to be suffering from any form of idiopathic fever. The man stated that he had had a bad cough for two months, and had kept his bed for two days. His breath had a most decidedly gangrenous odour; sputa of a dirty greenish muco-purulent character, and extremely fetid. Dry bronchial râles audible over chest, and at right base slight dulness, with increased vocal resonance, and large moist râles, but nothing approaching to cavernous breathing. Pulse 96; respirations 36. No change took place in physical signs of chest; but tongue became dry and brown; diarrhœa supervened; and patient gradually lost flesh and strength until death on Sept. 11.

On post-mortem examination, there was lobular pneumonia of lower lobe of right lung, and quite at base a gangrenous portion about size of an orange. Lung was here firmly adherent to diaphragm and diaphragm to liver, and the broken-down tissue of the gangrenous lung communicated by several openings with a cavity in upper part of right lobe of the liver, measuring about three inches in diameter. This cavity contained much calcareous matter and a quantity of a dirty greyish, very fetid, pultaceous substance. On careful examination, no hooklets of echinococci could be discovered. Rest of liver and the intestines healthy.

The absence of hooklets may be thought to negative the opinion that the tumour of the liver was originally an hydatid. But though these hooklets resist the changes which occur in the interior of the body for an indefinite period, they do not resist the putrefactive changes resulting from exposure to atmospheric air, and such exposure must have existed here for many weeks before death. An obsolete abscess is the only other lesion that could have produced the appearances described, but the man had never suffered from the symptoms of abscess of the liver.

In the two following cases, and also in Case XXXIX., the tumour appeared to compress the ureters.

CASE XLII.—*Hydatid of Liver—Pyelitis—Pus in Urine—Sudden Death.*

Ellen C——, aged 21, came under my care as an out-patient at Middlesex Hospital, in April 1861. She stated that for about eighteen months she had been getting very weak and losing flesh, and that latterly she had suffered from dyspnœa. She had no cough, but her father had died of consumption. She had also suffered from irregular menstruation and leucorrhœa. On examining chest, there was found to be a bulging of right side, commencing at upper border of fifth rib, attaining its maximum at false ribs, and then as gradually declining. Hepatic dulness in right mammary line extended for 3 in. below margin of ribs, and its total length was 6½ inches. The bulging below ribs occupied right hypochondrium and epigastrium, and extended over to left hypochondrium; it was slightly tender, and presented an elastic, almost fluctuating consistence, and on percussion communicated to finger the peculiar sensation known as 'hydatid vibration.' These characters were most marked in epigastrium. Superficial veins about epigastrium and hypochondrium much enlarged. Movements of respiration mainly confined to left side of chest. On right side, respiratory murmur could not be heard below fourth rib in front, or below lower angle of scapula posteriorly. Above this breathing was harsh and expiration prolonged. On left side dulness and absence of respiration up to within half an inch of lower angle of scapula. Patient could give no information as to length of time tumour had existed. In fact, she was quite ignorant of existence of any unusual swelling until it was pointed out to her. Her complexion was slightly sallow, but she had never suffered from jaundice or vomiting, and her bowels were regular; appetite very bad. In addition to tumour on right side, a painful swelling, apparently the displaced left lobe of liver, could be felt in left lumbar region in situation of kidney, and there was a copious discharge of pus in urine.

The patient remained under my observation for nearly twelve months. The dimensions of the tumour did not alter much, but, on the whole, they became slightly larger. From time to time she suffered severe pain in swelling in left lumbar region. At these times urine was clear, or almost so, and relief was always attended with a sensation of bursting and a return of pus in large quantity. Urine was repeatedly examined with microscope, but no pus, casts, or trace of echinococci could be discovered.

The treatment—which consisted in administration of tonics and iodide of potassium, and external application of iodine—failing to give relief, patient was admitted into hospital on Jan. 14, 1862, with the object of having a puncture made into the tumour in right hypochondrium. After remaining in hospital for six weeks she refused to give her consent, and was discharged at her own request.

I did not see the patient after this: but I ascertained that, on Nov. 6, 1863, she was admitted into University College Hospital, under care of Dr. Hare, to whom I am indebted for the particulars noted while she was under his observation. Towards the end of 1862 she had first suffered from pain in region of tumour in right hypochondrium. The pain was intermittent in its character, ceasing after a few days. For this she had been treated at the Female Hospital in Soho Square. The dimensions of tumour noted in University College Hospital showed that it had increased considerably. Although right costal angle was still greater than left, there was bulging of ribs on both sides as high as nipple, and dulness on percussion up to third rib on right side, and up to third intercostal space on left side. The heart was displaced upwards, its apex beating in third left intercostal space. Vertical hepatic dulness in a line with right nipple was 11¼ in., in mesial line 9½ in., and in a line with left nipple 9¾ in. Distinct fluctuation could be felt in epigastrium over a space measuring 4½ in. transversely and 2½ in. vertically; but there was now no hydatid fremitus. No œdema of legs. Patient was sallow; her urine contained no bile-pigment, but was still loaded with pus. She still suffered from attacks of pain in region of left kidney, which were always relieved by a sensation of bursting and a copious discharge of pus in urine. On admission, there was a considerable amount of pain and tenderness in region of tumour near umbilicus. This pain recurred from time to time, but was always relieved by leeches, poultices, and morphia. Patient also had an attack of pain and stiffness in left groin and knee, accompanied by enlargement of lymphatic glands in groin, and slight œdema in upper part of thigh. On Jan. 26, 1864, it was noted that she was free from pain, but that she had lost flesh and strength. On Feb. 9 she was discharged for unruly conduct.

The patient was confined to bed after leaving hospital, and died rather suddenly and unexpectedly at end of ten days. An hour before death she seemed tolerably well, and the probability is that the fatal event was due to the bursting of a hydatid cyst.

CASE XLIII.—*Hydatid Tumours of Liver and Peritoneum, compressing Ureters, and causing Degeneration of Kidneys.*

Mary Ann W——, aged 45, adm. into Middlesex Hosp. Dec. 15, 1864, under care of Dr. H. Thompson, and died Jan. 15, 1865. For a year before death she had suffered from headache and impairment of mental faculties, and seven weeks before death she had a fit of unconsciousness, followed by right hemiplegia, involuntary evacuations, and bed-sores. There were no symptoms referable to liver.

The arteries at base of brain were atheromatous, and there was an apoplectic cyst, with a patch of white softening in left corpus striatum. Liver, spleen, and diaphragm were adherent by fibrous bands. In the adhesions between spleen and liver was a cyst the size of a walnut, filled with soft putty-like matter, and lined by portions of a gelatinous hydatid membrane. In right lobe of liver was another cyst, the size of a small cocoanut, partly embedded in its substance and partly projecting from its upper surface, where it was firmly adherent to diaphragm. Its outer wall was partly calcified, and its interior was full of fragments of secondary gelatinous cysts and soft putty-like matter. Secreting tissue of liver healthy. In folds of mesentery of small intestine were three partly calcified cysts, varying in size from a hazel-nut to a walnut, and containing putty-like matter and secondary cysts. Greater part of pelvis was occupied by another large cyst, situated behind and above uterus, which was forced down so as to appear at vulva. This cyst contained a clear fluid and innumerable small cysts, varying in size from a pea to a walnut, all of them gelatinous and filled with a clear fluid. Another cyst, not so large, in right side of pelvis. Ureters were compressed by these cysts, and pelves of kidneys somewhat dilated. Kidneys small and granular, and cortices wasted and hardly distinguishable from cones. All of cysts in abdomen contained hooklets of echinococci.

In the following case secondary hydatid cysts were formed in the omentum and peritoneal cavity.

CASE XLIV.—*Hydatid Cysts of Liver and Peritoneum—Ascites and Anasarca of Lower Extremities—Albuminuria—Death.*

Catherine C——, a hawker, aged 45, was a patient in Middlesex Hospital from Jan. 10, 1865, until her death on June 21. With exception of an attack of rheumatism, she had enjoyed good health until about a month before admission, when she had been seized with violent pain in abdomen and loins, and at same time her legs and abdomen had begun to swell. While in hospital, she had ascites and great anasarca of lower extremities; urine contained albumen. She was treated with diuretics and purgatives, and her legs were punctured.

On examination of body, legs were very œdematous, and abdomen was greatly distended. Both lungs very œdematous, and right lung firmly adherent and carnified at its base.

Peritoneal cavity contained upwards of a gallon of clear serum, floating about in which were six nearly transparent hydatid cysts, with tremulous gelatinous walls, the largest about size of a hen's egg, and smallest about that of a walnut. The fluid in the floating cysts had a specific gravity of 1010, and contained no albumen; that in peritoneal cavity had a specific gravity of 1020, and was highly albuminous. Left lobe of liver partly atrophied, and between it and spleen, and firmly adherent to both and to stomach was a hydatid cyst the size of a fœtal head, containing a little clear fluid and innumerable smaller cysts of various sizes pressed together. In great omentum were three or four similar cysts the size of chestnuts, and attached to right kidney was another cyst as big as an orange. Echinococci were found in the larger cysts. Both kidneys much enlarged and fatty.

Case XLV. shows how closely hydatid of the liver may simulate cancer.

CASE XLV.—*Hydatid Tumour of Liver simulating Cancer—Discharge of Hydatids per anum, and temporary recovery.*

On Nov. 1, 1871, I saw, in consultation with Dr. Mackintosh, of Brompton Road, Mr. C——, aged 33, jobmaster, but formerly a publican. His illness was believed to have commenced only three or four months before with an attack of pleurisy on right side attended by effusion. Since then had lost flesh, and, two weeks before I saw him, abdomen had begun to swell. Never had syphilis; had not been intemperate, and no history of cancer in family. Over lower four-fifths of right chest there was dulness continuous with that of liver, and absence of breath-sound and of vocal thrill. Heart displaced to left, but no bulging of right side, and measurement of two sides equal. Moderate ascites. Lower margin of right lobe of liver, three or four inches below ribs, distinctly nodulated and tender. Occasional vomiting, but no jaundice.

Four days after this feet began to swell, and œdema rapidly increased; and on Nov. 8, when I saw patient a second time, temp. 101°, and pericardial friction over heart.

I did not see patient again, for, soon after my second visit, he was removed to Brighton. Here, I am informed by Dr. Mackintosh, he passed a large quantity of hydatid cysts per anum, the dropsy disappeared, and he got so much better that he was able to return to his employment in London. He died, however, about a year afterwards, while under the care of another medical man.

CASE XLVI.—*Hydatid Tumour of Liver—Secondary Hydatid Tumours in Spinal Canal—Paraplegia.*

The preparation of this case is in the museum of Middlesex Hospital (v. 15), and the following particulars are extracted from the Catalogue:—

'Vertebræ with spinal cord from dorsal region. The canal and dura-mater laid open. The pleura is separated from the ribs, and the sides of the bodies of the vertebræ by two hydatid cysts, one on each side. The hydatids have been opened in sawing through the laminæ of the vertebræ; but their walls remain, and the spinal cord is at this place considerably smaller than elsewhere.

'The patient was a woman aged 40, who had been admitted into the hospital with paraplegia and retention of urine. She died with a large slough on the sacrum, and the bladder was found to be inflamed. There was also a large hydatid cyst in the liver.'

In the following case a process of spontaneous cure appears

to have commenced in the tumour, and the observatio
teresting in connection with the manner in which a
probably effected in a hydatid tumour, when the fluid
are drawn off by means of a small trocar and cannula (s
73 and 83).

CASE XLVII.—***Large Hydatid Tumour of Liver, full of sec***
Cysts, but containing no Fluid.

This liver was taken from body of a man, aged 36, who
mitted into the Fever Hospital on Dec. 2, 1866, with hæn
small-pox, of which he died on Dec. 5. He was too ill to
particulars of his previous history.

After death, a hydatid tumour, the size of a child's head, w
in posterior part of right lobe of liver. The chief points of
in the case were that this cyst was tightly packed with second
and that it contained no fluid. The secondary cysts were c
but still they exhibited their natural gelatinous appearanc
were not at all opaque or mixed up with any putty-like mater
outer cyst, however, at several places presented an atheromat
fied appearance.

CASE XLVIII.—*Large Hydatid of Liver undergoing 'spontane*
from calcification of cyst, and discovered after forty-five ye

On Feb. 10, 1873, I was requested by Dr. W. Steer Ridi
Mr. W——, aged 56, on account of a remarkable tumour
with the liver. Liver did not ascend too high in front or at b
lower margin of right lobe descended to two inches below u
and the portion below ribs felt as hard as bone, and was sm
painless. Patient had no symptoms referable to tumour, and
an active life, until a trifling ailment of lungs led to discover
Riding of tumour, as to existence of which patient himself
rant. He remembered, however, that when a child, at least
before, he had been brought a long distance from the co
London to see Sir Astley Cooper and another surgeon; that
had then been said to be four times its proper size, and
thought to contain fluid, and that there had been a questio
forming an operation. It had been decided not to interfere
tumour had gradually got smaller as he had grown older.

LECTURE IV.

ENLARGEMENTS OF THE LIVER.

CONGESTION—INTERSTITIAL HEPATITIS—INFLAMMATION OF BILE-DUCTS—OBSTRUCTION OF COMMON DUCT.

Gentlemen,—In the previous lectures I have called your attention to the distinguishing characters of the four enlargements of the liver which are for the most part unattended by pain. Those in which pain is a prominent symptom remain to be considered. Seven diseases are included under this head; viz. 1. congestion of the liver; 2. interstitial hepatitis; 3. inflammation of the bile-ducts; 4. obstruction of the common duct and retention of bile; 5. pyæmic abscesses; 6. tropical abscess; 7. cancer. Speaking generally, it may be said that jaundice, which is a rare symptom in painless enlargements of the liver, is present to a greater or less extent in the class of enlargements now to be noticed; tropical abscess is the one in which it is oftenest absent. Ascites is also a common symptom. First among the enlargements of the liver attended by pain comes—

V. CONGESTION OF THE LIVER.

In the first place, it is necessary to bear in mind in reference to the pathology and treatment of this condition, that the quantity of blood in the liver varies greatly at different times consistently with health, and that even these healthy variations may influence to some extent the size of the organ. For instance, the amount of blood in the liver and its size are greatly influenced by diet, both being temporarily increased after a meal, and particularly when the food has been too large in quantity, or has contained an excess of fatty, saccharine, or alcoholic ingredients. By morbid congestion of the liver, we mean something more than this. The phrase 'congestion of the liver' is *too often* used very vaguely, and applied to cases of indigestion where there is probably little amiss with the liver. True congestion of the liver is distinguished by the following characters :—

1. There is enlargement of the liver which is uniform in character—not greater in one direction than in another—and which is rarely very great. The liver may project an inch or more below the margin of the ribs in the right mammary line. In the venous engorgement from mechanical obstruction of the circulation, the enlargement is usually greater than in active congestion, where the engorgement commences in the arteries. Another peculiarity of this enlargement is that it is rarely permanent, but that after a time it usually disappears. Even when the cause of the congestion is most permanent, such as mechanical obstruction of the cardiac circulation from valvular disease of the heart, the enlargement of the liver gives place after a time to an opposite condition of contraction. The pressure exerted by the constantly distended hepatic veins causes atrophy of the central portions of the lobules, and induces a form of granular liver, different from true cirrhosis, where the atrophy commences at the circumference of the lobules.

2. The surface of the portion of liver projecting below the ribs is smooth.

3. The patient complains of a feeling of tightness or painful distension in the region of the liver, and there is more or less—but rarely very acute—tenderness on pressure below the margin of the right ribs. The pain and feeling of uneasiness may, in consequence of the connection of the subclavius nerve with the phrenic, stretch up to the right shoulder, and they are almost always increased after meals or by lying on the left side. In the latter case there is usually a sense of dragging or weight in the hepatic region. The patient consequently sleeps for the most part on his back, or on his right side.

4. Jaundice is present in most cases after two or three days, but is rarely intense, and it is not often that bile is altogether absent from the motions. When there is intense jaundice with absence of bile from the stools, catarrh of the ducts is probably present, as well as congestion of the hepatic tissue.

5. There is usually nausea, with loss of appetite, headache, furred tongue, a bitter taste in the mouth, flatulence, and other symptoms of indigestion, and not unfrequently there is vomiting or diarrhœa, or both. The same cause that produces congestion of the liver may induce a similar condition of the stomach and

intestines; slight irritation then suffices to induce catarrhal inflammation of the mucous membrane of these parts, of which vomiting and diarrhœa are the prominent symptoms. With these derangements of digestion it is not uncommon to find anæmia, general languor and debility, emaciation, depression of spirits, drowsiness, and hypochondriasis.

6. More or less dyspnœa is not uncommon, even in cases where the primary disease is not in the chest, and many patients are harassed by a frequent dry cough. The dyspnœa may be so great as to raise the suspicion of serious mischief in the heart or lungs, but it is often entirely removed by free purgation.

7. Signs of obstructed portal circulation are not uncommon. In acute cases there may be tension in the left hypochondrium, and an increased area of splenic dulness; while in more protracted cases there may be hæmorrhoids or ascites.

8. The urine is usually scanty and high-coloured, and besides containing more or less bile-pigment, often deposits a copious sediment of lithates or lithic acid. Temporary albuminuria is not uncommon.

9. As in other forms of enlargement of the liver, the circumstances under which the enlargement appears constitute an important aid to the diagnosis of the real nature of the case. Hepatic congestion may be mechanical, active, or passive, and the chief conditions under which it occurs are the following:—

A. *Mechanical.*—Among the most common causes of hepatic congestion in this country is mechanical obstruction of the circulation in the chest, and particularly that consequent on disease of the mitral or tricuspid valves of the heart. In many cases of valvular disease of the heart, a time arrives when the chief symptoms are those of hepatic congestion, and the main treatment must be directed to their relief.

B. *Active.*—Several causes contribute to the development of active congestion:—

a. Irritating ingesta, in the form of alcohol, fermented liquors, spices, or food which errs in being habitually too rich in quality or in excessive quantity may cause congestion of the liver. The temporary increase of blood in the liver always present after a meal may become morbid in degree and permanent, if the ingesta be habitually of an irritating character. Congestion of the liver is more likely to result from these causes in weakly persons who lead indolent and sedentary lives, than in persons of a robust constitution who take plenty of muscular exercise in the open air.

b. A high temperature is usually reckoned among the causes of congestion of the liver, but probably rarely leads to such a result except in conjunction with irritating ingesta. It is to this combination of causes that must be attributed the frequency of active congestion of the liver among Europeans in warm climates. (See Lect. XVI.)

c. A sudden or protracted chill may induce congestion of the liver, especially in warm climates, in persons who have been free livers, or after violent exercise.

d. Malaria and Blood-Poisons.—Persons who suffer from malarious fevers, or live in malarious districts, are very prone to have congestion of the liver, which may persist long after the febrile symptoms have passed away. Officers and soldiers not uncommonly return from India with enlargement of the liver from this cause. But when great and permanent enlargement of the liver succeeds to ague or remittent fever, it is more probably the result of waxy deposit, or of interstitial hepatitis, than of simple congestion.[1] There are other blood-poisons, besides malaria, which may induce congestion of the liver, such as the yellow fever of the tropics, and the relapsing fever of our own country.

e. Active congestion of the liver may have a traumatic origin, and result from contusions, wounds, &c.

C. *Passive.*—Passive congestion of the liver may be due to:—

a. Suppression of habitual discharges, as of the catamenia, or of the bleeding from piles. I have repeatedly known congestion of the liver, and even cirrhosis, follow a successful operation for piles.

b. Habitual constipation.

c. Torpor of the portal vascular system from paralysis of the sympathetic nerves or from any other cause.

d. Insufficient muscular exercise.

Treatment.—In the treatment of hepatic congestion, you must be guided by the following rules:—

1. In all cases it is well to commence by removing, if possible, the cause. The measures to be adopted for this object will be apparent from what has already been stated.

2. In most cases of any severity advantage will be derived

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[1] See page 36 and Case X., and also Morehead, Res. on Dis. in India, 1860, p. 428; and Sir Ranald Martin, in Lancet, 1865, ii. p. 615.

from the employment of local depletion in the form of leeches or of cupping to the region of the liver, or, what is still better, the application of a few leeches around the anus. If depletion be deemed inexpedient, sinapisms may be applied over the liver. After the leeches or the sinapisms, their place ought to be supplied by linseed or bran poultices. Tepid baths are sometimes useful.

3. The diet should be of the least irritating character. Only small quantities of milk, beef-tea, or farinaceous articles ought to be taken at a time. Alcohol, wine, fermented liquors, spices, fat, and all rich or indigestible articles ought to be rigidly interdicted. In modern practice much mischief is often done by compelling patients with heart-disease and congestion of the liver to swallow large quantities of brandy.

4. Purgatives are in most cases of great utility, unless there be spontaneous diarrhœa, which ought not to be too speedily or completely checked. Purgatives in fact are the best means of checking the frequent, but fruitless, calls to stool from which the patient often suffers. The best purgatives are those salines which increase the watery exhalation from the mucous membrane of the bowels, such as the sulphates of magnesia, potash, and soda, the tartrate of potash and soda, seidlitz powders, Carlsbad salt, and Friedrichshall or Püllna water. These salts ought to be dissolved in warm water and taken in the morning on an empty stomach. Their action is often materially assisted by an occasional dose of calomel, blue-pill, or podophyllin, which bring away copious bilious motions.[1]

5. When the congestion is traceable to irritating ingesta, an emetic in the early stage sometimes appears to do good, by clearing out the stomach and duodenum. The pressure also to which the liver is subjected during the act of vomiting may squeeze out of it some of the superfluous blood.

6. During the persistence of the symptoms of congestion—enlargement and tenderness of the liver with jaundice—and especially in those cases where there is much gastric derangement, alkalies and their salts with the vegetable acids ought to be prescribed. They may be taken two or three times a day

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[1] The increased biliary excretion after the calomel in these cases is not due to an increased secretion of bile by the liver, but probably to the mercury acting upon the upper part of the small intestine, so that the bile is propelled onwards, instead of being reabsorbed (see Lect. IX). If calomel acted by stimulating the liver to increased secretion, it would be *injurious* in cases of hepatic congestion.

shortly before meals. The alkaline mineral waters, such as those of Vals, Vichy, and Ems, or the artificial effervescing Vichy salt, may often be advantageously substituted for the alkaline preparations of the Pharmacopœia.

7. The chloride of ammonium has been found to be of great utility in hepatic congestion in this country as well as in India.[1] In doses of twenty grains two or three times daily, it induces free diaphoresis, increases the flow of urine, diminishes portal congestion, and relieves hepatic pain. It is believed also to stimulate the absorbents, especially those in the liver, and thus to effect the absorption of hepatic abscess. It may be given in combination with either alkalies or acids.

8. Ipecacuanha has been recommended by Dr. C. Maclean[2] as one of the best and safest remedies in the acute hyperæmia of the liver, which in tropical climates is so often the precursor of suppurative inflammation. He believes it to be a blood-depurant; it increases the secretion of the liver and skin, and so there can be no doubt as to its beneficial action in the cases referred to. It is a notable fact that since ipecacuanha has come into general use in the treatment of dysentery in India, abscess of the liver has become much less frequent. As in dysentery, it must be given in large doses (20 to 30 grains) every six or twelve hours according to the severity of the case. Quarter of a grain of tartar emetic and 15 grains of nitrate of potash, given every half-hour until the pain is relieved, is said to act in a similar manner.

9. When the more urgent symptoms have passed off, and the patient suffers chiefly from debility, anæmia, and dyspepsia, with a slight increase of the hepatic dulness, with or without hypochondriasis, the treatment may be modified. The mineral acids and vegetable tonics are now often useful, such as the mineral acids with taraxacum, nux vomica, or gentian. Quinine and iron are particularly indicated in patients who have suffered from malarious fevers; but ought to be given with great caution to persons of gouty habit, or who have been free livers. The diet ought also to be more generous, although care must be taken to exclude from it every source of irritation. Fermented liquors ought still to be interdicted, and if wine be

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[1] Although this drug had been long used in various hepatic disorders, its value in the treatment of hepatic congestion was first made known in 1869 by Dr. William Stewart, of H.M. Army.

[2] Reynolds's System of Med. iii. 337.

allowed at all, it should be given in small quantities, and diluted. Hock, claret, and dry sherry are the best. Regular exercise in the open air ought to be enjoined; if there be much debility, the advantages of exercise without fatigue may be derived from riding on horseback. The bowels will still require attention, and great benefit will often be obtained from the use of mineral waters which combine chalybeate with purgative properties, such as the springs of Harrogate, Cheltenham, Leamington, Homburg, and Kissingen.

10. It is in the chronic condition last referred to that advantage is sometimes derived from the use of the nitro-muriatic acid bath, as recommended by Sir Ranald Martin.[1] The bath should consist of two ounces of strong hydrochloric and one ounce of strong nitric acid to two gallons of water, at a temperature of 96° or 98°. Both feet are to be placed in the bath, while the inside of the legs and thighs, the right side over the liver, and the inside of both arms, are sponged alternately, or the abdomen may be swathed in flannel soaked in the fluid. The process is to be continued for half an hour night and morning.[2] In obstinate cases advantage is sometimes derived from the hydropathic belt, or from inunction with the ointment of biniodide of mercury.

As an example of congestion of the liver resulting from mechanical obstruction of the circulation in the chest, I may call your attention to the following case:—

CASE XLIX.—*Mitral Stenosis—Dropsy and Congestion of Liver—Death.*

Emma F——, aged 13, adm. into Middlesex Hosp. Oct. 24, 1865, suffering from much cough, great dyspnœa, and considerable anasarca of lower extremities. Cardiac dulness had double its normal area, and a prolonged bellows-murmur was audible over left apex. There were all the signs of general bronchitis; and, in addition, conjunctivæ and general surface had a slightly jaundiced tint; hepatic dulness was much increased, measuring in right mammary line more than 5 in. and extending down nearly to umbilicus. Splenic dulness also increased. Considerable tenderness below right ribs. Tongue furred. Much nausea and occasional vomiting, and bowels relaxed about four or five times a day; motions pale, though coloured with bile. Urine contained a small amount of bile-pigment, but no albumen. Five or six years before, this patient had an attack of scarlet fever, followed by articular rheumatism and dropsy. Ever since, she had suffered from dyspnœa and palpitations, increased by any exertion. About ten days before admission she began to complain of cough, headache, and vomiting, and swelling appeared in ankles, which gradually extended upwards.

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[1] See Lancet, Dec. 9, 1865, p. 641.

[2] The bath, as above prepared, may be kept in use for a few days, 1 drachm of hydrochloric and half a drachm of nitric acid, with a pint of water, being added daily to make up for waste. About a fourth of the fluid is to be well heated in an earthen pipkin, so as to bring up the temperature of the whole to 96° or 98°. Glazed earthen or wooden vessels should be used, and the sponges and towels should be kept in cold water, lest the acid *corrode them.*

The treatment consisted in administration of purgatives and diuretics, and particularly the bitartrate of potash and tincture of digitalis, while leeches and mustard and linseed poultices were applied over right hypochondrium. At first there was a manifest improvement in all the symptoms; but about a fortnight after admission the indications of obstructed cardiac circulation became aggravated; dyspnœa and dropsy increased, lips and face were livid: jaundice was more marked, vomiting more urgent, and motions contained less bile. Pulse was very rapid, and on Nov. 10 scarcely perceptible. At 11 P.M. of this day the girl died.

On examination of body, heart was much enlarged, weighing 13 oz.; mitral valve much thickened and its margins adherent, so that orifice was contracted, and its circumference measured only fifteen lines. Both lungs much congested, and presented the ordinary anatomical characters of bronchitis; but they were nowhere consolidated. Peritoneum contained about a pint of clear serum. Liver very large for patient's age, weighing nearly 4 lbs; outer surface smooth; and, on section, roots of hepatic vein gorged with dark blood, contrasting strongly with intermediate pale-yellow hepatic tissue. On microscopic examination, quantity of oil in secreting cells did not seem increased. Spleen weighed 6¼ oz., and was firm and dark on section. Pyramids of kidneys much congested, but renal tissue in other respects healthy. Mucous membrane of pyloric half of stomach presented ordinary characters of catarrhal inflammation.

As an illustration of congestion of the liver arising from other causes I may narrate to you the following case:—

Case L.—*Indigestion from Habitual Surfeit—Residence in Tropics—Exposure to Chill—Congestion of Liver.*

Mr. C——, aged 30, a gentleman much addicted to the pleasures of the table, consulted me in June 1867, on his return from India. He had for several years suffered from constipation, flatulence, and a feeling of weight and oppression in region of liver. About six weeks before I saw him, he was attacked with pain in region of liver followed by vomiting and jaundice, after sleeping on a verandah in the night air in India. He had leeches applied over liver, and was ordered home at once. I found him still moderately jaundiced; liver enlarged, measuring 5 in. in right mammary line, and slightly tender; no vomiting, but bowels constipated; a bitter taste in mouth, and nausea. Motions light but contained bile. Urine scanty, dark, contained bile-pigment, deposited much lithates, and became very dark on addition of nitric acid after heating. He was treated with saline purgatives and occasional pills of the comp. colocynth mass (gr. vi), podophyllin (gr. ⅓), and extract of henbane (gr. ii); an effervescing mixture of citrate of potash was ordered to be taken three times a day; a warm bath three times a week; moderate exercise; and a simple diet, from which alcohol in every form was excluded. At the end of ten days patient was much improved, jaundice had almost gone, and hepatic dulness diminished. A mixture with nitric acid and compound infusion of gentian was now substituted, and in two or three weeks more patient had regained his usual health.

VI. ENLARGEMENT OF LIVER FROM INTERSTITIAL HEPATITIS.

This form of enlargement of the liver is a common sequel of chronic hyperæmia. Its clinical characters are these:—

1. The enlargement is uniform in every direction, and may be much greater than in simple congestion. The liver may reach up to the nipple and down to the navel, or even lower, but its lower margin is often obscured by tympanites or ascites.

2. Its surface is smooth, or slightly uneven, dense and resisting, and more or less tender. Occasionally there is acute tenderness from intercurrent attacks of peri-hepatitis.

3. The symptoms in the first instance are the same as those of active hyperæmia, which I have already described to you, so that sometimes it may be difficult to say whether there is congestion only, or congestion plus interstitial hepatitis.

4. But when the disease is more pronounced, its prominent features are sallowness or slight jaundice, venous stigmata on the cheeks, nausea and retching, especially on first rising in the morning, loathing of solid food, particularly in the early part of the day, diarrhœa alternating with constipation, hæmorrhoids, scanty dark urine loaded with lithates, and in some cases temporary albuminuria, depression of spirits, sensations of sinking and a craving for stimulants. Occasionally there is slight pyrexia.

5. In a still more advanced stage there will be the various symptoms of portal obstruction, which I shall have to describe to you in detail when we come in a future lecture to consider

cirrhosis under the head of contractions of the liver. The enlargement in fact which I have now described is the disease known as cirrhosis, although when the symptoms of this disease are well pronounced the liver is more commonly contracted. In opposition to the opinion of the late Dr. Todd,[1] several observers, such as Saunders, Bright,[2] Budd, Frerichs[3] have expressed the opinion that in cirrhosis the contraction of the liver is occasionally preceded by a stage of enlargement, but as far as can be judged from medical writings such an enlargement is believed to be exceptional. Gee has recorded two cases of 'cirrhotic enlargement of the liver,' in which the organ weighed 100½ and 104 ounces respectively.[4] Habershon has met with an inflammatory induration of the liver, in which the organ becomes greatly enlarged;[5] and Duckworth has described a 'hypertrophic cirrhosis.'[6] From my own experience I have been led to believe that in a considerable proportion of cases of cirrhosis, the liver is still much enlarged (very often from the presence in the organ of a large quantity of fat) after ascites and other symptoms of portal obstruction have set in, and that patients often die in this condition with jaundice, hæmorrhages, and symptoms of blood-poisoning (the prognosis being no better than if the liver were contracted). In this opinion I am confirmed by the independent observations of Professor Leudet, of Rouen,[7] who observes: 'On est arrivé par l'anatomie pathologique à reconnaître que l'augmentation du volume de la glande n'était pas toujours l'indice d'une lésion récente du foie, d'un processus aigu encore curable.' This is the reason why I have brought the disease under your notice on the present occasion, but I shall have occasion to return to it in a future lecture. It is a matter for investigation, whether, if the patient lived long enough, the enlargement in all these cases would be followed by marked cirrhotic contraction. M. Ollivier, in fact, is of opinion that cirrhotic enlargement is a distinct affection from cirrhotic contraction.[8] There can be no doubt, however, that it occurs under the same conditions and gives rise to the same symptoms. It seems probable, therefore, that the same causes sometimes lead to contraction, and sometimes to enlargement of the liver.

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[1] Clin. Lect. on Urinary Diseases and Dropsies, 1857, p. 113.

[2] Guy's Hosp. Rep., 1st ser. vol. i. p. 612.

[3] Dis. of Liver, Syd. Soc. Transl. vol. ii. pp. 35, 37, 53.

[4] St. Barth. Hosp. Rep. 1869, vol. v. p. 108.

[5] Lettsomian Lectures, 1872, p. 56.

[6] St. Barth. Hosp. Rep. vol. x.

[7] Clin. Méd., Paris, 1874, p. 541.

[8] L'Union Méd. Sept., 1871, pp. 361, 400, 449.

6. The causes of interstitial hepatitis may be said to be these:

a. In a large proportion of the cases presenting the clinical features which I have now described, you will find that the patient has been addicted to a free use of alcoholic drinks. You must beware of being deceived in this matter. Such patients may tell you, and even really believe, that they lead regular and temperate lives, because they never drink a sufficient quantity of alcohol at one time to obscure their intellects; but it is the practice of 'nipping'—of taking frequently small quantities of spirits, or a glass of sherry, under the mistaken notion that this better fits them for work,—that keeps the liver in a constant state of congestion, and most surely leads to cirrhosis. Moreover, you must not gauge one man's capacity for alcohol by that of another. One person may take with impunity, what in another will induce serious disease.

b. The congestion of the liver which results from venous obstruction may also lead to an interstitial hepatitis presenting most of the clinical characters which I have now described (Case LV.), but distinguished from true cirrhotic enlargement by the presence of chronic cardiac or pulmonary disease, and of the signs of obstructed systemic circulation. In rare cases I have known the liver from this cause not only enlarged, but nodulated.

c. Interstitial hepatitis resulting in enlargement of the liver may also have a syphilitic origin, although more commonly in these cases the liver seems to contract from the first. These cases will be distinguished by the history of constitutional syphilis, and by the greater tendency to attacks of severe perihepatitis causing much pain and tenderness. The liver also has a greater tendency to become uneven or nodulated from the cicatrix-like depressions which form upon its surface, or from the projection of enlarged and softened gummata. In the latter case the disease may be mistaken for abscess[1] or hydatid; and indeed, from what was observed in Case LVII. and in a case recorded by Dr. Moxon[2] where a syphilitic gumma in the liver softened into a puriform fluid and burst into a bile-duct, it seems not improbable that a tumour of this sort may occasionally discharge itself by the stomach or bowels. In other cases there may be a difficulty in distinguishing the disease in question from waxy enlargement with peri-hepatitis.

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[1] Wilks, British Med. Journ. 1876, i. 239.

[2] Path. Trans. vol. xxiii. p. 153.

d. Lastly, a chill, independently of intemperate habits, would appear to be in rare instances sufficient to excite interstitial hepatitis ending in great enlargement of the liver. Dr. Wilson Fox has communicated to me the particulars of such a case, where there was a persistent though slight elevation of temperature, and I have observed one or two similar instances, in which, however, the diagnosis was not, as in Dr. Fox's case, verified by post-mortem examination. It is probable that in these cases some constitutional dyscrasia predisposes to the action of the chill. (See Appendix, Case CLXXVI.)

The treatment of interstitial hepatitis in its early stage will be the same as that which I have already indicated as appropriate for congestion; that for the advanced stages will be more conveniently discussed when I come to speak of cirrhotic contraction. Syphilitic cases will of course call for specific remedies, and especially for mercury and iodide of potassium.

The four following cases are examples of cirrhotic enlargement of the liver resulting from alcohol. The first three are illustrations of the good effects of treatment; while Case LIV. illustrates the appearances found after death.

CASE LI.—*Cirrhotic Enlargement of Liver from Alcohol (and Malaria?) —Great Ascites—Paracentesis—Recovery under treatment.*

On Jan. 5, 1873, I saw, in consultation with Dr. A. Simpson, of Highgate, Mr. L., aged 35, an indigo-planter, just returned from India, where he had been born, and where he had lived all his life. Had been a free liver and drunk much spirits, but excepting several attacks of malarious fever had good health until June 1872, when he began to have considerable hepatic pain, followed by ascites and swelling of legs. Girth at umbilicus on Jan 5, 37½ in.; much fluid in peritoneum; great œdema of legs. Liver large, projecting nearly 4 in. beyond right ribs, very hard and distinctly nodulated. Urine had contained albumen, but was now free from it. Bowels confined. Face sallow, with venous stigmata.

On supposition that patient might be suffering from effects of malarial cachexia, iron, quinine, and strychnia were prescribed, with an aperient draught every morning containing sulphate of magnesia and iodide of potassium. Dropsy, however, increased. On Jan. 13 girth of abdomen 38¾ in.; legs more swollen; and penis and scrotum very œdematous. On Jan. 26 girth of abdomen 41 in. and respiration embarrassed. A mixture of digitalis, bitartrate of potash, and juniper was now substituted for the quinine and iron, and the purgatives were continued. Abdomen was also fomented with a strong infusion of digitalis. On Feb. 22 the mixture was changed for one of perchloride of mercury and digitalis. Under this treatment at first slight and temporary improvement, but on March 3 girth of abdomen 42½ in.; signs of fluid in lower fourth of both pleural cavities; orthopnœa; urine only 30 oz. Ordered podophyllin pills and a mixture of digitalis, squill, and juniper. On March 5 about two gallons of fluid were drawn off by paracentesis, with immediate relief. At first ascites seemed to be collecting again, and on March 8 girth of abdomen 38 in.; but after this swelling of abdomen slowly receded, and on March 31 girth only 32 in.; no fluid in pleuræ. Liver still reached down to umbilicus, hard and nodulated. Purgatives were continued, and a pill three times a day containing gr. ⅛ of green iodide of mercury was substituted for diuretic mixture, and red iodide of mercury ointment was rubbed in over liver. The iodide of mercury was continued for two months, dose being gradually increased to gr. ½. Under this treatment he steadily improved. On April 29 he was able to drive four miles to my house. On May 30 girth still 32 in., but no sign of ascites, no œdema of legs, and liver a little smaller. He was now ordered nitro-muriatic acid, bark and taraxacum, with aperients, and he went to Devonshire. From this time there was no return of the dropsy, and he steadily improved; in November he weighed more than ever he had done in his life. On March 17, 1874, liver scarcely exceeded normal dimensions. In October 1874 he returned to Tirhoot in India, where he remained until following April; and on June 11, 1875, when I last saw him, he was still in good health, free from dropsy, and liver of about natural size.

CASE LII.—*Cirrhotic Enlargement of Liver—Ascites—Good Effects of Treatment.*

On March 29, 1873, I was consulted by Capt. M., aged 40, of R.N., on account of enlargement of liver and ascites. He was a short spare man, had been long on the Indian seas, and had drunk freely of brandy. About a year before, when in China, he began to have pain in liver and morning sickness and diarrhœa; and at end of November 1872 abdomen began to swell, until when he left China in February its girth was 38 in., and the legs were also swollen. When I saw him girth was reduced to 32 in. but still much ascites; liver 8 in. in r. m. l., of which four inches below ribs. Spleen hard, and somewhat tender, also much enlarged. Heart sound. No albuminuria. Face sallow, with venous stigmata.

Ordered to abstain from stimulants; Carlsbad salt every morning; blue pill, squill, and digitalis twice daily; and a mixture of iron and nitrous ether; and on April 9 ordered to rub red iodide of mercury ointment every night over liver. Under this treatment he slowly improved, and on April 15 no fluid in peritoneum; girth of abdomen 29 in.; liver in r. m. l. 7½ in. During summer he drank waters at Homburg for five weeks, and on Aug. 20 liver in r. m. l. 6¼ in., and could walk several miles without any œdema of legs. From this time he felt fairly well until October 1874, when he had again uneasiness about liver with loss of appetite and diarrhœa. For these symptoms he consulted a medical man, who prescribed astringents and opium with port wine, and after a fortnight abdomen again swelled. On Dec. 14 much ascites and enlargement of abdominal veins; girth of abdomen 34½ in.; legs œdematous; bowels costive; occasional retching; liver 6 in. Port wine was at once stopped; Carlsbad salt ordered every morning; and a mixture of iron, bitartrate of potash, and digitalis. Ascites at first increased, and on Jan. 7 girth nearly 37 in. A mixture of perchloride of mercury and digitalis was now substituted for the iron &c. Under this treatment, modified somewhat from time to time, great improvement again took place; urine became very copious; and on May 6, girth 29 in., no ascites, and liver in r. m. l. 6 in., of which 2 in. below ribs. He again went to Homburg for five weeks, and on his return on July 14, 1875, he appeared to be in excellent health, without any sign of dropsy, but liver still 6 in. in r. m. l. and hard.

Case LIII.—*Cirrhotic Enlargement of Liver.—Ascites and Albuminuria—Weak Heart—Good effects of Treatment.*

On April 15, 1873, I was consulted by Mr. James V., aged 56, on account of disease of liver and dropsy. He was a large corpulent man, who had been a free liver, and had drunk much wine and spirits. Seven years before, he had an attack of congestion of liver and had passed much blood per anum. For years he had had occasionally slight swelling of legs, but one month before I saw him abdomen began to swell, after which legs increased rapidly. For six months before abdomen began to swell, had suffered from dyspeptic symptoms and despondency. Liver very large (8 in. in r. m. l.) hard and uneven; much ascites; girth 48 in.; enormous swelling of penis and scrotum; great œdema of legs, with numerous large ulcers; urine contained ¼ albumen and hyaline casts; heart's sound weak, but no bellows-murmur.

Stimulants were restricted to a pint of hock or claret daily; patient was ordered a black draught with jalapine every morning, and a mixture of bitartrate of potash, squill, and digitalis. Under this treatment, modified from time to time, and with occasional courses of iron, great improvement took place. Urine became copious and free from albumen; ascites and dropsy of legs disappeared; and liver was reduced. On June 3 girth of abdomen only 40½ in., and on July 29, 39½ in. For many months after this, patient enjoyed good health and went about town, although he was less prudent than he ought to have been. There was occasionally a return of albuminuria, and once or twice urine contained much sugar and specific gravity rose to nearly 1040; but usually urine contained neither sugar nor albumen, and specific gravity was under 1020. In July 1874 there was a return of ascites, and girth of abdomen rose to 44 in.; it again disappeared under similar treatment, and on Oct. 18 girth only 40 in. In January 1876 there was again a slight and temporary return of ascites, brought on apparently by imprudence in diet. In August 1876 he had another more severe attack of ascites and dropsy of legs; but under use of elaterium and diuretics this completely disappeared, and by middle of October patient was walking about London, with a good appetite and free from dropsy and albuminuria, but with a large hard liver still reaching almost to umbilicus.

CASE LIV.—*Large smooth Cirrhotic Liver simulating Waxy Disease—Ascites—Persistent Diarrhœa—Death.*

Elizabeth R——, aged 40, adm. into St. Thomas's Hosp. Sept. 11, 1875. Nothing remarkable in family history. Married; five living children from 16 to 5; three miscarriages, first 12 months after marriage, a three months fœtus; last, four months before admission. General ichthyosis, but no history of syphilis. Habits not very temperate; admitted to 1½ pt. beer daily, besides some spirits; had suffered from morning sickness independently of pregnancies. Nine months before admission, vomiting became more frequent; had great pain across stomach and in back, with persistent diarrhœa, cough, loss of appetite and flesh, and abdomen began to swell.

Remained in hospital until Oct. 25, and during this time condition was as follows:—Emaciated; venous stigmata on cheeks; jaundiced tint of conjunctivæ; abdomen enlarged, measuring 39 in. at umbilicus; liver much enlarged, measuring 9 in. in r. m. l., and lower margin felt hard, sharp, and even, below umbilicus; surface smooth; moderate ascites; frequent vomiting and constant diarrhœa; no sign of cardiac disease, but slight dulness and tubular breathing at apex of right lung; temp. at night usually 101°; occasional attacks of profuse epistaxis. No albuminuria. On leaving hospital, seemed better; no ascites and no diarrhœa.

One month after leaving hospital abdomen began to swell again. Towards end of January 1876 diarrhœa returned, and on Feb. 29 patient was readmitted into hospital. Her condition was as follows. Very weak and emaciated; girth of abdomen at umbilicus 37½ in.; liver still very large, measuring in r. m. l. 9½ in., and extending from nipple to below umbilicus; surface hard and slightly tender, generally smooth, but a large projecting mass felt in epigastrium: spleen enlarged; moderate ascites; constant diarrhœa, 8 or 10 watery motions, without pain, daily; no vomiting; tongue unnaturally red and clean;

appetite bad. Sallow and anæmic; no decided jaundice, but urine contained bile-pigment and $\frac{1}{8}$ albumen. Almost constant epistaxis and great fetor of breath. Pulse 120. Heart healthy.

Patient was ordered milk diet, and a mixture of bismuth and opium, and subsequently pernitrate of iron, but she became daily worse. On March 2 she vomited about a pint of dark blood; the ascites diminished slightly, but the diarrhœa and epistaxis persisted, and on March 15 she died from exhaustion.

Autopsy.—Six pints of ascitic fluid in peritoneum. Liver very large, weighed 74 oz., firmly adherent to transverse colon, stomach, &c. Capsule greatly thickened, and on upper surface two loculated cysts of ascitic fluid; lower margin rounded; typical cirrhotic structure on section; no amyloid reaction. Spleen 12$\frac{1}{2}$ oz., congested. Kidneys firm, but yielded no amyloid reaction. Right lung adherent.

Case LV. appeared to be an example of great enlargement of the liver from interstitial hepatitis consequent on mitral disease.

CASE LV.—*Great Enlargement of Liver and Ascites, secondary to Mitral Disease.*

Edwin F——, aged 11, adm. into St. Thomas's Hosp. Nov. 3, 1871. Had enjoyed good health till last July, when he was laid up for several weeks with a severe attack of rheumatic fever, and since then he had suffered from palpitations and dyspnœa. On admission heart greatly enlarged, measuring 3$\frac{1}{2}$ in. transversely, its apex beating between 6th and 7th ribs outside nipple. At apex was a loud whistling systolic bellows-murmur, heard also at lower angle of left scapula and in fact all over chest. Pulse 108, small and weak. Occasional cough, but lungs healthy. Liver slightly enlarged. Slight œdema of legs. Albumen ($\frac{1}{4}$) in urine.

Was ordered digitalis and iron, and on Nov. 9 appearance greatly improved, and albumen disappeared from urine.

On Dec. 14, while still in hospital, was seized with a second attack of articular rheumatism, which became complicated with pericarditis and pleuro-pneumonia. For several weeks he was extremely ill, and the disappearance of the pericarditis and pneumonia was followed by a great aggravation of the cardiac symptoms. On Jan. 22 transverse dulness of heart 4 in., breath very short, much cardiac pain and palpitation; considerable œdema of legs and some ascites, but no albumen in urine. Diuretics and iron were of little use, and on March 18 both legs, which were enormously enlarged, were punctured with considerable relief. On April 1 fomentation of abdomen with infusion of digitalis four times the Pharmacopœia strength had the effect of increasing flow of urine and reducing dropsy. After a few days, however, dropsy again increased in abdomen, until, on April 22, girth at umbilicus was 33 inches, but there was little or no œdema of legs. Liver was greatly enlarged, extending from right nipple to umbilicus, its surface smooth, hard, and slightly tender; abdominal veins enlarged; no jaundice; much albumen in urine; dyspnœa urgent. By paracentesis abdominis 172 oz. of fluid were now drawn off, with great and immediate relief. The albuminuria at once ceased; and under use of digitalis with other diuretics, blue pill, purgatives, and subsequently iron, ascites did not again collect; liver diminished somewhat in size, and cardiac symptoms improved. On Aug. 5 left hospital free from dropsy, and girth at umbilicus only 25 in.

Was again a patient in hospital from Nov. 13 to Dec. 5, 1872, with albuminuria, slight ascites, (abdomen measuring 27¾ in.) but no œdema of legs. Under use of blue pill, digitalis, diuretics, purgatives, and iron, albuminuria and ascites again completely disappeared; and boy, on leaving hospital, went to sea-side. Liver was still large.[1]

Cases LVI. to LXI. are examples of syphilitic enlargement of the liver with gummata.

In Case LVI. the syphilitic nature of the disease in the liver was not suspected during life. The concurrence of great enlargement of the spleen, persistent diarrhœa, copious albuminuria without general dropsy, and great anæmia, suggested that the enlargement of the liver was due to waxy disease, and the ascites was referred to compression of the portal vein by lymphatic glands enlarged from waxy deposit. The profuse catamenial discharge was, however, the only cause that could be assigned for waxy disease.[1]

CASE LVI.—*Syphilitic Hepatitis and Gummata of Liver—Waxy Spleen—Ascites—Diarrhœa—Jaundice.*

Sarah B——, aged 25, was a patient in Middlesex Hosp. from April 21 to June 2, 1868, for anæmia, enlargement of liver and spleen, albuminuria, ascites, and diarrhœa. Since first appearance of catamenia at age of 12, when she had copious flooding, she had suffered from anæmia and chlorosis, and she had been much worse since her marriage in 1866. She had never been pregnant, and after most careful enquiry nothing could be elicited pointing to a syphilitic history. Her father had died at 40 of effects of an accident: her mother and one sister had died of consumption. At commencement of 1866 abdomen had begun to swell and diarrhœa set in. At time of admission girth of umbilicus was 34½ in.; hepatic dulness in r. m. l. rose to nipple and measured 4¼ in.; vertical splenic dulness 6 in.; urine contained ⅕ albumen; bowels open ten to twelve times a day. Heart elevated but healthy. Under the use of nitric acid and opium, diarrhœa ceased; albumen was reduced to a mere trace, and ascites disappeared, although abdomen still measured 33 in.

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[1] See also Case LXXXIX. p. 488, in 1st edition.

Was again a patient in Middlesex Hosp. from Nov. 2 to Dec. 1, 1868. Had then slight jaundice, distinct ascites; liver 5 in. in r. m. l.; spleen projected 5 in. beyond ribs; 6 stools daily; no albumen in urine during whole time; but systolic murmur at base of heart. Was again relieved by same treatment as before.

Was a third time a patient in hospital, from July 9 to Aug. 10, 1869. Still ascites and slight jaundice. Liver dulness 5 in. and spleen 5 in. beyond ribs. Girth of abdomen 35½ in. Urine contained a trace of albumen; 8 to 10 stools daily; menorrhagia; anasarca of legs. Under same treatment diarrhœa again ceased, and patient gained flesh and strength.

Was a fourth time a patient in hospital with same symptoms from Nov. 23, 1869, to Jan. 8, 1870. Girth of abdomen 36 in.; 7 or 8 stools; albumen $\frac{1}{20}$.

Soon after leaving hospital on Jan. 8, diarrhœa returned and abdomen became larger. Came several times as an out-patient, and on March 17, 1870, was admitted for a fifth time. Girth of abdomen was now 43 in., and corresponding to umbilicus was a protrusion as large as an orange, integuments of which were red, thin, glistening, and tender; but abdomen generally not tender. Superficial veins of thorax and abdomen much enlarged. No appetite; much flatulence; three or four stools daily; occasional retching. Considerable dyspnœa; resp. 48, and thoracic. Pulse 108; no bellows-murmur with heart. Urine contained fully one-half albumen and some bile-pigment; no casts. Slight œdema of legs. Marked chlorosis. No jaundice. All treatment on this occasion proved useless. Patient became rapidly worse. On March 21 passed very little urine, was restless and wandered; on 22nd unconscious; on 23rd pulse intermittent and diarrhœa increased. On 24th she died.

Autopsy.—No visible cicatrices on vulva or on vagina, but a deep cicatrix on anterior lip of uterus. Peritoneum contained 100 oz. of clear yellow serum, with a few flakes of lymph; entire membrane presented signs of recent peritonitis, vessels being intensely injected and intestines plastered with soft yellow lymph. Firm adhesions between liver and diaphragm and right kidney, &c. Liver rather small, its capsule thickened and its outer surface marked by numerous deep cicatrix-like depressions, and on cutting into several of these they were found to be connected with characteristic syphilitic gummata, some as large as cherries. Hepatic tissue pale and friable, fatty, and with no amyloid reaction. Much fibrous tissue in portal fissure, compressing but not obliterating portal vein. Considerable hypertrophy of connective tissue in interior of liver. Round ligament much thickened. Spleen 30¼ oz.; capsule much thickened; its tissue firm and waxy, with distinct amyloid reaction. Kidneys large and pale (7 oz. each), with amyloid reaction of small arteries. No ulceration and no amyloid reaction of intestines.

CASE LVII.—*Syphilitic Enlargement (Gummata) of Liver—Gummata in one arm—Periostitis of one tibia.*

On July 28, 1875, I was consulted by Mrs. R——, aged 37, on account of a tumour of liver, regarding which different opinions had been expressed by the many medical men whom she had seen. Some had said that it was hydatid; others, abscess; others, cancer; and one distinguished physician had pronounced it an adenoid tumour. The liver was very large, extending from nipple to navel, and portion below liver bulged forward and was very soft and elastic but painless; its surface was distinctly nodulated, one nodule in epigastrium very like cancer, most prominent parts being the softest; there was jaundice, which came and went, and was sometimes attended by white stools; no ascites; spleen much enlarged, projecting four inches beyond ribs. No albuminuria. Slight œdema of legs, and some periostitic swelling of left tibia, not of right. Temp. normal, appetite good. History was this:—Mother had died of cancer of uterus. Married 16 years; no children; one doubtful abortion six weeks after marriage, but with this exception never pregnant. More than four years ago began to have a vaginal discharge, for which, on medical advice, she took much mercury and was salivated. Two years ago noticed first one, and then a second, small lump below right ribs in front. These lumps had increased steadily from first, but more rapidly of late.

Although it was not supposed that the tumour contained fluid, it was determined to remove all doubt by making an exploratory puncture. This was done, but only blood escaped. Nitro-muriatic acid, nux vomica, and saline aperients were ordered. Under this treatment she felt better and stronger, and there was no increase of swelling. On Oct. 31 she vomited a little blood, and for several days afterwards she had much nausea and was deeply jaundiced. About this time first observed a lump about size of half an orange in soft parts of left upper arm. This increased slightly and became red and soft in centre, and very painful, and for this I was again consulted on Jan. 18, 1876. Iodide of potassium was prescribed, but two days afterwards patient had an attack of very profuse hæmorrhage from stomach and bowels, and medicine was suspended. Swelling in arm increased, and on Feb. 7 a slough, size of half-a-crown, had formed in centre of lump, circumference of which was still very hard and tender. Iodide of potassium was resumed (gr. iij ter die). On Feb. 28, slough separating; a distinct periostitic node on left tibia; no return of hæmorrhage; liver apparently not larger than six months before; girth over most prominent part 34½ in.; appetite good, but painful distension after food. No albumen in urine. Was ordered iodide of potassium, ten grains, with sarsaparilla, three times daily. Under this treatment, alternated occasionally with small doses of perchloride of mercury and bark, patient greatly improved. On March 27 slough had separated from arm and sore was healing, jaundice less, and girth only 33¾ in. On May 2 sore in arm quite healed, leaving deep cicatrix; but severe periostitic pains in right ulna and fingers, and in right tibia. On June 1 periostitic pains gone; scarcely any jaundice; up, and going about. July 19: has driven out five or six times; gaining flesh; girth 35 in.; severe periostitic pain over right elbow. July 20: something seemed to burst inside, and she vomited a quantity of yellowish matter, which was not preserved. Had nausea for several days after, but then continued to improve. Sept. 26: liver decidedly smaller; girth only 33½ in., although she has grown very much stouter. Spleen not diminished in size. No jaundice; appetite good. Still has periostitic pains in right ulna preventing sleep.

Case LVIII.—*Syphilitic Enlargement of Liver—Gumma in left leg.*

Mr. J——, about 47, consulted me for first time on May 4, 1874. About 16 years before he had syphilis. The sore was slight, and he does not remember having had constitutional symptoms. Six years afterwards he married; his wife had no children or miscarriages. In 1871 he began to feel stuffed up in nose, and soon after he noticed an offensive discharge, with occasional clots of blood, from nose. The discharge also passed backwards. After ten months a piece of bone, size of sixpence, came away from right nostril and discharge ceased. About same time gums were in a very bad state and he had five teeth drawn. Soon after this he got better, and remained well until Nov. 1873, when one day after lunch he got squeamish, had pain in region of liver with slight jaundice, and for five weeks was unable to go to business. Ever since he had suffered from flatulence and other symptoms of indigestion, and also from piles; but, although he had been in habit of drinking a good deal of whisky, he never had nausea or morning sickness, and had always a good appetite for breakfast. There was dilatation of capillaries of cheeks, but no jaundice. Liver very large, measuring 8 in. in r. m. l.; enlargement uniform; smooth, hard, and painless. Spleen also somewhat enlarged; no ascites. Tongue preternaturally clean and red; bowels usually costive. Urine 1024, usually loaded with lithates, but free from albumen. Pulse 96; heart normal.

He was ordered to abstain from stimulants, except a little claret and water, to take a dose of Carlsbad salt every alternate morning, and a mixture of perchloride of mercury and chloride of ammonium three times a day. Under this treatment urine became copious and clear, symptoms greatly improved, and liver was slightly reduced in size. On Oct. 24 it measured only 7¼ in. in r. m. l., but there was a small painful ulcer on left tonsil, and often in morning a little blood was discharged from nose. After this he took iodide of potassium occasionally, but he always felt better while taking the perchloride of mercury. In Dec. 1874 he first noticed a lump in middle of left leg over tibia, but quite unconnected with bone. This increased to size of an egg and softened; in May 1875 it was opened; no pus, but much clotted blood, escaped. Wound was many months in healing, and patient was unable to walk much on account of pain in left ankle. On healing, a deep scar remained, covered by a scab, and surrounded by considerable induration. In Feb. 1876 this was seen by Sir James Paget, who at once pronounced it to be a syphilitic gumma. Liver still large, but measurement in r. m. l. not more than 6½ in.; surface smooth. Still has pain about left ankle which prevents him walking much, but appetite and digestion are good. He was ordered a mixture of perchloride of mercury with bark; and in October (1876) I heard that his health had greatly improved.

CASE LIX.—*Enlarged Fibrous Syphilitic Liver with Gummata—Ascites.*

Harriet R——, aged 28, adm. into St. Thomas's Hosp. Feb. 1, 1875. Nothing noteworthy in family history. Had enjoyed good health until three years before admission, when she had for some weeks an obscure attack of pain and swelling in lower part of abdomen; but after this got quite well and married in Jan. 1873. One miscarriage, but no living child. Early in 1874 began to suffer from dry cough, flatulent distension of abdomen, and retching from slight causes; but no morning sickness, and habits temperate. In November 1874 abdomen became swollen and painful; and since then excessive vomiting brought on by sight or smell of food; increase of cough; loss of appetite; emaciation; and attacks of abdominal pain, sometimes so severe as to keep her in bed for a week.

On admission, emaciated and countenance expressive of pain; no jaundice or venous stigmata on cheeks; pain and tenderness of abdomen, which measured at umbilicus 43½ in. Much ascites; abdominal veins but slightly dilated; neither liver nor spleen could be felt, but upper margin of hepatic dulness reached almost to nipple; tongue too clean and red; frequent retching; bowels costive. Urine contained phosphates, but free from lithates or albumen. Pulse 96, small and feeble; apex of heart elevated, but no abnormal murmur. Frequent dry cough; respiration thoracic and somewhat laboured; but lung signs normal. Slight pitting of legs. Temp. 99°.

Was treated with purgatives and diuretics, including digitalis; but as no improvement resulted, and patient was suffering great pain and distress of breathing from abdominal distension, paracentesis was performed on Feb. 6, and 19 pints removed of straw-coloured serum;

sp. gr. 1016. Operation gave great relief. After removal of fluid, upper margin of hepatic dulness still nearly reached nipple. Lower margin of liver could be felt projecting more than three inches beyond ribs in r. m. l., hard, indented, and tender, but surface smooth. Girth 36 in. On Feb. 10 was ordered iodide and citrate of potash, with digitalis and decoction of broom-tops; and for this on Feb. 20 a mixture of perchloride of mercury, squills, and digitalis was substituted. Fluid gradually re-accumulated, and on Feb. 24 girth 43 in.; urine scanty; paracentesis; 18 pints drawn off; sp. gr. 1015. Operation again gave immediate relief, and on both occasions was followed by great increase in flow of urine. From March 3 to 13 had erysipelas of face and scalp spreading to abdomen, during which pulse rose to 160, temperature to 104°, dry tongue, and much delirium. After this extreme prostration, large abscesses, one containing more than a pint of pus, formed beneath skin on different parts of body, and girth of abdomen increased to 45½ in. On April 2, five pints, and on 9th, 24 pints of fluid (sp. gr. 1015) drawn off by paracentesis. Died from exhaustion on April 27.

Autopsy.—Peritoneum contained 24 pints of serum. Liver enlarged; weighed 62 oz.; firmly adherent to all adjacent organs; capsule thickened; substance indurated from interstitial hepatitis; numerous firm syphilitic gummata, some as large as cherries, mainly distributed along course of portal vein and of its branches, some of them forming projections from surface of liver. Portal vein in fissure of liver much dilated. Spleen 14 oz.; firm. No amyloid reaction in liver, spleen, or kidneys. Recent pleurisy on both sides; both lungs œdematous.

VII. INFLAMMATION OF THE BILIARY PASSAGES.

This condition is usually associated with more or less congestion of the hepatic tissue, and accordingly its clinical characters are those of congestion, which I need not recapitulate, with those peculiar to catarrh of the bile-ducts and gall-bladder superadded. Thus we have—

1. Enlargement of the liver, which, like that from congestion, is uniform in every direction, and rarely very great; but which is sometimes accompanied by enlargement of the gall-bladder in the form of a more or less pyriform tumour projecting from the anterior margin (see fig. 16, page 163). In some cases no enlargement can be made out.

2. The portion of liver projecting below the right ribs is smooth on palpation.

3. There is at first a feeling of tightness and distension in the right hypochondrium, with tenderness on pressure, particularly

over the enlarged gall-bladder. Sometimes, however, there is little or no pain or uneasiness. The pulse is usually abnormally slow.

4. Inasmuch as the bile-ducts are obstructed from the tumefaction of the mucous membrane, as well as from the inflammatory products thrown off from its free surface, the jaundice, after a day or two, is much more intense than in simple congestion, and the motions contain no bile.

5. Here, again, the circumstances under which the attack occurs are of great assistance in diagnosis.

a. In a large majority of cases the attack is preceded by symptoms of catarrh of the stomach and duodenum. The inflammation, in fact, commences in the mucous membrane of the digestive canal, and extends thence to the common bile-duct. Accordingly there are to be noted, in the first place, a furred tongue, loss of appetite, flatulence, nausea or vomiting, pain and tenderness at the epigastrium, and sometimes diarrhœa, these symptoms being often accompanied by slight pyrexia. After a few days or longer, jaundice appears, and the fever, if present, may subside, although the dyspeptic symptoms remain. Attacks of this sort are very common in children as the result of eating indigestible food, or of a surfeit; and, in that case the jaundice and other symptoms usually subside at the end of ten days or three weeks.

b. Catarrh of the bile-ducts (like catarrh of the bronchial tubes) is not uncommon in persons of more advanced age of a gouty constitution, and more than once I have met with cases of this description, where the frequent vomiting, the emaciation, and the jaundice persisting for many weeks, have led to the suspicion of cancer, but have soon subsided under the use of purgatives with colchicum and alkalies.

c. Catarrh of the bile-ducts is one of the diseases of the liver consequent on syphilis. The jaundice which is not uncommon during what is known as the secondary stage is usually due to this cause, and it is especially in cases of this nature that acute atrophy of the liver is apt to supervene.

d. Inflammation of the biliary passages may be secondary to congestion or other diseases of the liver, and then its symptoms may be persistent. It is probable that catarrh of the ducts may not only excite congestion of the hepatic tissue, but may result from it. In any case, where congestion of the liver is developed under the circumstances already mentioned, and where, in addition to the symptoms of simple congestion, there is intense jaundice, with an absence of bile from the motions, we may infer that there is catarrh of the ducts as well as congestion. Other diseases of the liver, also, such as the waxy liver and hydatid tumour, are occasionally complicated with catarrh of the ducts; and in this way jaundice may appear in the course of diseases of the liver in which it is usually absent. In another lecture (Lect. VII.) I shall have occasion to mention an example of enlargement of the liver from tubercular deposit where the jaundice was apparently due to inflammation of the common bile-duct.

e. Inflammation of the bile-ducts and gall-bladder may be due to the irritation of gall-stones or of other foreign bodies. Under these circumstances it will usually be distinguished by a previous history of biliary colic, which, however, was notably absent in the case of one patient who lately died in the wards (Case LXV.).

f. Certain poisons, such as those of pyæmia and phosphorus, have been stated by Virchow to excite catarrh of the bile-ducts.[1] The cause of that form of catarrh of the bile-ducts known as 'epidemic jaundice' is probably some poison contained in the air or in drinking water.

Speaking generally, it may be said that in young people catarrh of the bile-ducts is the most common cause of jaundice; whereas in persons of middle or advanced life, if we can exclude syphilis and a gouty habit, jaundice is probably due to some other cause than catarrh.

Treatment.—The rules already laid down for the treatment of congestion of the liver are also applicable to catarrh of the bile-ducts. Little more need be added except that—

1. Leeches and cupping are less necessary in simple catarrh. In most cases sinapisms and warm fomentations, with purgatives, alkalies, and chloride of ammonium, suffice for subduing the disease. The propriety of employing local depletion must be decided by the degree of pain and amount of congestion existing in each case.

2. When there is reason to suspect that the affection is of a gouty nature, great benefit will often be derived from the addition of colchicum and iodide of potassium to the remedies already mentioned. In these cases also it will be necessary to adopt such measures as are calculated to correct that disordered condition of the digestion, which, if neglected, will before long lead to a recurrence of the hepatic attack.

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[1] Virchow's Arch. 1865, xxx. Hft. 1.

3. In syphilitic cases the most useful remedies are the perchloride of mercury and chloride of ammonium, in conjunction with purgatives.

4. The treatment must occasionally be modified by the presence of other diseases of the liver, of which the catarrh of the bile-ducts is merely a complication.

When I come to lecture on Jaundice, I shall have to return to the subject of catarrh of the bile-ducts (Lect. X); but in the meantime I would direct your attention to the following cases of painful enlargement of the liver accompanied with jaundice, apparently due to catarrh of the ducts.

CASE LX.—*Painful Enlargement of Liver, with Jaundice due to Catarrh of Bile-ducts.*

Elizabeth L——, aged 21, a maid-servant, was admitted into Middlesex Hosp. on Dec. 7, 1866. For nine months she had been weakly and unable to take a place, and had also suffered from dyspeptic symptoms. Ten days before admission, at the cessation of last catamenial period, which had its usual duration, she had been seized with great nausea and vomiting, but she had no diarrhœa. Five days after this she began to complain of pain and tenderness in region of liver, but pain was never very severe. About same time jaundice made its appearance, which increased in intensity, and was accompanied by much itchiness of skin.

On admission, deep jaundiced colour of entire skin and conjunctivæ; urine very dark, and gave characteristic reaction of bile-pigment; tongue thickly coated; no appetite, but vomiting and pain in side had much subsided; lower margin of liver was ascertained to project about an inch below margin of ribs in right mammary line, and here there was slight tenderness on pressure; bowels rather constipated; motions clay-coloured, without a vestige of bile-pigment; pulse 100; skin rather hot (temp. 100° F.); respiration slow and easy; physical signs of heart and lungs normal.

The treatment consisted in the frequent administration of saline purgatives (sulphate of magnesia), and a blue pill occasionally at bedtime, together with the application of mustard and linseed poultices to region of liver.

Bowels were freely purged, and on Dec. 17 the symptoms had considerably improved; pulse had fallen to 68; tongue clean; neither nausea nor vomiting; appetite returning; urine contained less bile-pigment. No change, however, had taken place in colour of skin and conjunctivæ, which were still deeply jaundiced. An alkaline mixture, containing bicarbonate of soda, chloric ether, and tincture of orange, was now substituted for the sulphate of magnesia; a purgative was still given occasionally, and patient had a warm bath twice a week.

On Dec. 20 jaundiced tint was first noticed to be giving way, and from this date it gradually faded until Jan. 7, 1867, when it had quite disappeared. A tonic mixture with nitric acid and quinine was now ordered, and on Jan. 22 patient left the hospital in good health.

The following cases are cited as illustrations of catarrh of the bile-ducts occurring in gouty individuals. (See also Case CXXI.).

Case LXI.—*Gouty Dyspepsia—Enlargement of Liver, and Jaundice from Catarrh of Bile-ducts.*

In autumn of 1865 I was consulted by Mr. C. D——, a gentleman aged 30. His father had been a martyr to gout, and a younger brother had suffered from it early in life. He had never had well-marked gout himself, but he had long been liable to gastric derangements characterised by nausea and flatulence and transient pains in small joints. About three weeks before I saw him he had been seized about an hour after dinner, with a pain at the epigastrium, followed by vomiting and nausea. A few days later jaundice appeared, and gradually increased in intensity; the nausea continued without vomiting, and patient became much emaciated. On examination I found lower margin of liver projecting more than half an inch beyond edge of ribs in right mammary line, and slightly tender on pressure; intense jaundice of a deep olive tint; great itchiness of skin, and complete absence of bile from motions. Urine dark, like porter. Pulse 60; no appetite, nausea and flatulence after everything swallowed. Patient was extremely weak and thin, and his appearance in an older man would certainly have suggested the existence of malignant disease of stomach or liver.

The treatment adopted consisted in application of mustard and linseed poultices to region of liver, warm baths, blue pill with saline purgatives, a mixture with citrate of potash and vinum colchici, and a diet restricted to milk, beef-tea, and farinaceous articles.

After two days symptoms began to improve, and by end of three weeks jaundice had quite disappeared and patient was restored to his usual health.

Case LXII.—*Gout—Catarrh of Bile-ducts—Jaundice.*

Alfred B——, 38, leather-cutter, adm. into St. Thomas's Hosp. Oct. 17, 1874. Six years before had left facial paralysis for twelve months. Ten years before, eldest brother (four years older) had gout, and patient himself had been in habit of drinking much beer. Five weeks before admission, wrists, fingers, ankles, and knees been swollen and painful. After about a week or ten days, pain and ing subsided, but skin and conjunctivæ became very yellow, and he had much itchiness of skin and occasional vomiting.

State on Admission.—Deep jaundice. Liver slightly enlarged, measuring 5 in. in right nipple line; lower edge smooth and painless. Tongue white; moderate appetite; much flatulent distension after meals; bowels costive; motions white; no vomiting for a fortnight. Urine clear, but loaded with bile pigment. Pulse 108. Heart and lungs sound. Temp. 101° F.

Treatment consisted of a mixture, three times daily, containing citrate of potash, iodide of potassium, and vin. colchici, and compound rhubarb and blue pills on alternate nights, followed by a black draught next morning, with milk diet. At the end of a week bile appeared in stools and jaundice began to fade. On Nov. 12 jaundice had quite disappeared, and two days later patient left hospital well.

In the two following cases catarrh of the bile-ducts with jaundice appeared to result from syphilis.

CASE LXIII.—*Constitutional Syphilis—Catarrhal Jaundice.*

Edwin R——, aged 25, adm. into Middlesex Hosp. Dec. 8, 1868.

Had good health until four months before, when he contracted primary syphilis, followed by enlargement of inguinal glands and a roseolar rash. Four weeks before admission, he began to suffer from nausea, occasional vomiting, pain about right shoulder-blade, a feeling of weight in head, dimness of sight, and general debility; a week later he became jaundiced, and had diarrhœa with much flatulence.

On admission, great weakness; jaundice of skin and conjunctivæ, and numerous copper-coloured spots of psoriasis upon skin. Tongue white; moderate appetite; pain in abdomen and eructation of gas after food; vomiting and diarrhœa ceased; no bile in stools. Liver enlarged, measuring 6 in. in r. m. l. and extending 2 in. beyond ribs; surface smooth and slightly tender. Pulse 84, regular, Temp. 100·1°. Urine 1020; no albumen, but much bile-pigment. Was ordered pil. coloc. co. with podophyllin, sulphate of magnesia and senna draughts, and a mixture containing potass. bitart. and sp. æth. nit.

Under this treatment the bowels and kidneys acted freely, but no material improvement took place; and on Dec. 16 jaundice not at all diminished, urine loaded with bile-pigment, and stools clay-coloured. The patient was now ordered liq. hydrarg. perchlor. ʒj ter die. On Dec. 21 there was decided evidence of bile-pigment in urine, and patient was ordered a warm bath and Dover's powder at bed-time. A few days later the jaundice began to fade; and on Dec. 30 there was only a trace of bile-pigment in the urine, the liver was reduced in size, and the jaundice had almost disappeared.

Case LXIV.—*Catarrh of Bile-ducts from Syphilis (or Arsenic?) Jaundice.*

Josephine S——, 29, nurse, adm. into Middlesex Hosp. May 30, 1871. Within three years had passed through attacks of small-pox, relapsing fever, and scarlatina. A widow; had borne four children, of whom one stillborn and another died within a month of birth. Denied syphilis, but six weeks ago had a sore throat which lasted for some days. On May 19 noticed an eruption on arms, neck, and chest, for which on 26th she consulted a doctor, who gave her a solution of arsenic, of which she was to take 5 drops three times a day. On May 27, after taking fourth dose of medicine, had great nausea, and next day after dinner, and also after medicine, she vomited and bowels acted four times. She now discontinued medicine, but on 29th she again vomited and complained of pain and tenderness about liver, and in the evening she became jaundiced. On morning of admission had vomiting and slight purging.

State on admission.—Decided jaundice. An eruption of elevated copper-coloured, scaly spots over arms, back, front of chest, and neck. Tongue moist, with white fur and red edges; much thirst; no appetite, motions clay-coloured. Has dull pain in right hypochondrium, with some tenderness below right ribs; liver projects about an inch beyond ribs in r. m. l. Urine 1025; much bile-pigment, but no albumen. Temp. 100-101·5°. Pulse 112; over third left intercostal space distinct roughness of first sound of heart.

Patient was ordered 6 grains of calomel, an occasional 'black draught,' and an effervescing alkaline mixture, and had mustard and linseed poultices applied over right side, while diet was restricted to milk, bread, and beef-tea. On June 3 the cutaneous eruption had increased, and patient complained of sore throat and a deep ulcer was discovered on right tonsil. This was touched with solid nitrate of silver, and mixture was changed for one containing iodide and bicarbonate of potash. On June 1 a little bile was observed in stools, but no material improvement took place until June 8, when bile was passed freely from the bowel. On June 9 no trace of bile-pigment could be found in urine, and after this jaundice rapidly faded. On July 4 patient was discharged free from jaundice, with ulcer of tonsil healed and eruption almost gone.

In the following case death was due to uræmia from diseased kidneys, but the hepatic symptoms appeared to result from inflammation of the gall-bladder and bile-ducts excited by gall-stones which was subsiding before death.

CASE LXV.—*Inflammation of Biliary Passages excited by Gall-stones—Gangrene of Foot—Diseased Kidneys—Death by Uræmia.*

Many of you will remember the patient J. K——, aged 49, who was a patient in Middlesex Hospital from Oct. 27, 1866, until his death on Nov. 21. His story was that he had enjoyed good health until the previous June, when he began to suffer from loss of appetite, lowness of spirits, and pain and flatulence after meals. About same time he got a rusty nail into his left big toe. This resulted in an abscess, which burst and continued discharging until a few days before admission. He had continued working, however, as a labourer until within the last three weeks. During his illness his weight had diminished from 12 st. to 11 st. 5 lbs. On Oct. 20 he had a severe rigor, lasting for three hours, and followed by a rather severe constant 'gnawing' pain, with tenderness in region of liver, vomiting of bitter green fluid, and headache. Two days later his skin became jaundiced, and he suffered from itchiness of skin and loss of sleep. About same time that jaundice appeared, left big toe became black, and the ulceration extended. At no time of his life had he suffered from symptoms of biliary colic.

On admission it was noted that patient had rather deep jaundice of skin and conjunctivæ. He complained of general itchiness, and of dull pain in region of liver, which was uniformly enlarged, dulness in right mammary line being 5½ in. There was also decided tenderness at a spot corresponding to gall-bladder, which was also enlarged. Abdomen distended and tympanitic; ingesta were constantly vomited within half an hour; tongue moist, jaundiced, and furred; bowels costive; motions clay-coloured. Urine of the colour of porter, and contained a large quantity of bile-pigment, and also of albumen, with granular and a few oil-casts. On dorsum and sole of left big toe were several large sloughy ulcers, the surrounding soft parts being much swollen and livid. Pulse 72; skin cool; there had been no rigors or perspirations. Patient was treated with blisters and mustard and linseed poultices to region of liver, while bismuth, chloric ether, purgatives, &c., were given internally.

For some time there appeared to be considerable improvement: jaundice diminished, and bile reappeared in considerable quantity in motions. But about Nov. 12 vomiting became more urgent, and prostration increased. On Nov. 19 left foot was found to be much swollen, and livid lines marking course of lymphatics passed up legs. On Nov. 20 an abscess was opened above left ankle, from which fetid pus and gas escaped. On same day the man was seized with a fit of convulsions, followed by coma. These fits recurred in rapid succession, so that he had nearly thirty before his death at five P.M. on Nov. 21.

On examination of body after death, brain and its membranes were found to be normal, except that there was a considerable amount of fluid, which contained urea, at base and in lateral ventricles. Kidneys enlarged, and much fatty and granular deposit in secreting cells. Liver large, weighing 80 ounces; secreting cells loaded with oil; lobules unusually distinct, giving a granular appearance to organ on section. Gall-bladder contained a soft black concretion as large as a walnut, and many small, irregularly-shaped fragments of same material. These were suspended in a small quantity of dark-green viscid fluid, which, on microscopic examination, was found to contain a large number of pus-corpuscles. Mucous surface of gall-bladder had a stretched, white appearance, and at fundus was deeply injected, granular, and excoriated. Bile-ducts contained a similar viscid fluid to that in gall-bladder, with minute particles of black inspissated bile. This could be squeezed into duodenum without much difficulty. Mucous membrane of stomach and duodenum minutely injected, with numerous small ecchymoses, and surface coated with much viscid mucus. Great œdema and congestion of both lungs. Fat was deposited in large quantity throughout body, and all the soft tissues were deeply jaundiced.

The next form of enlargement of the liver, attended by pain and jaundice, to which I wish to direct your attention, is—

VIII. ENLARGEMENT FROM OBSTRUCTION OF THE COMMON BILE-DUCT BY CALCULI, TUMOURS, ETC.

Obstruction of the common bile-duct may lead to enlargement of the liver in two ways.

a. By causing dilatation of the biliary passages with accumulation of bile in them. It is not uncommon to find the ducts larger than the middle finger, and many instances are on record where the dilatation has been even greater than this.

b. By inducing inflammation of the biliary passages associated with more or less congestion and an overgrowth of the connective tissue. The liver in these cases is of a deep bilious or olive-green colour, and its consistence is increased. It must not, however, be forgotten that, if the obstruction be of long standing, the liver may ultimately contract to less than its natural size, its secreting tissue becoming atrophied from the pressure of the distended bile-ducts and of the newly formed connective tissue. On microscopic examination the secreting cells are found to be reduced in size and very often to contain an undue amount of oil, and in cases of long standing they may be completely destroyed: while in the capillary bile-ducts bile may sometimes be seen crystallised in the form of irregular, ruby-red, shining bodies, differing in form from crystals of hæmatoidin. The primary enlargement is usually followed by atrophy in about three or four months, but the time varies in different cases.

The distinguishing characters of the enlargement of the liver that occurs under such circumstances are as follows:—

1. The enlargement is rarely great, and, with one important exception, it is uniform in every direction. The exception referred to is due to the enlargement of the gall-bladder, which can often be felt as a pyriform tumour projecting from the lower margin of the liver. This enlargement is due, in the first place, to an accumulation of bile, but after a time not unfrequently to the admixture or substitution of inflammatory products. The late Dr. Bright has recorded a case in which such an enlargement of the gall-bladder formed an oval tumour descending nearly to the crest of the ilium; and you have had an opportunity of examining a similar, though smaller, tumour in the case of J. W——. (Case LXVI. and fig 16.)

2. There is jaundice, which if the cause of obstruction be a gall-stone, like the pain about to be referred to, is often in the first instance paroxysmal, but by the time that the liver becomes enlarged is permanent and usually intense, and is accompanied by a total disappearance of bile-pigment from the motions. In cases of persistent jaundice, where from the colour of the motions it is clear that the flow of bile into the bowel has been cut off for many weeks, there can be little doubt that there is obstruction of the common duct; and if the jaundice has been preceded by paroxysmal pain, the cause of that obstruction is probably an impacted gall-stone. But if there be no evidence of the jaundice having been preceded by paroxysmal pain, it may be difficult to say whether the obstruction be due to an organic obliteration of the duct at its duodenal opening from an ulcer or from a cancerous growth in the duodenum, or to a tumour in some other part of the course of the duct, or to pressure by a tumour on the duct from without. The rules for your guidance under these circumstances will be best considered when I come to describe the various forms of jaundice arising from obstruction of the common bile-duct.

3. Pain and tenderness in the region of the liver, and particularly in the situation of the enlarged gall-bladder, are present in most cases. The pain is greatest in those cases where there is peri-hepatitis, or cancer of the liver, or where the bile-duct is compressed by a tumour which at the same time compresses and stretches the hepatic plexus of nerves. When the obstruction is due to the impaction in the duct of a gall-stone,

there will be a history of attacks of paroxysmal pain with the other phenomena of biliary colic, but all pain may have ceased before the patient comes under observation.

4. The diagnosis will usually be assisted by the presence of those symptoms which mark the various morbid conditions producing obstruction of the bile-duct, and which will be considered hereafter under the head of Jaundice.

The *treatment* of this form of enlargement of the liver, or rather of its various causes, will also be best considered under the head of Jaundice.

In the meantime, I may recall to your recollection the following case, which has been under your observation for some weeks, and which is a good illustration of enlargement of the liver and jaundice, apparently from gall-stones, except that the patient's age is considerably under that at which gall-stones are ordinarily met with. The enlargement of the gall-bladder and many of the other symptoms appeared to be due to catarrhal inflammation of the bile-ducts and gall-bladder, excited by a gall-stone.

Case LXVI.—*Enlargement of Liver and Dilatation of Gall-bladder from Obstruction of Common Duct by a Calculus.*

John W——, aged 30, a stone-cutter, adm. into Middlesex Hosp. Feb. 5, 1867. He had enjoyed good health until six months before, when he began to suffer from acute paroxysms of pain in abdomen. For a week he would have several paroxysms daily; then he would be free for a week, and during this interval he would be able to resume his work. The attacks were not accompanied by vomiting, but the first was followed by jaundice, which had never left him. The paroxysms continued to recur for six weeks, but subsequently to this he had none; he had suffered much, however, from flatulence and itchiness of skin, and had lost flesh. On admission, universal jaundice of moderate intensity: urine loaded with bile-pigment, but motions contained none. Hepatic dulness moderately and uniformly increased, measuring five inches in right mammary line. No tumour corresponding to gall-bladder could be discovered, but possibly this was obscured by the flatulent distension of bowels; no ascites. Tongue moist, and but slightly furred; appetite good, and no vomiting; but patient was obliged to be very careful as to diet, as he suffered much from flatulence and pain after eating; pulse 72.

About a fortnight after patient's admission he became much worse; and on Feb. 20 it was noted that jaundice was more intense, urine darker, and hepatic dulness increased, measuring fully 5½ in. in right mammary line. In addition, there was now in situation of gall-bladder a distinct tumour (see fig. 16), extending 1½ in. below margin of liver, measuring 2½ in. transversely, and tender on pressure. Temperature had risen to 104·2° F., and pulse to 96. Tongue somewhat dry, motions perfectly devoid of bile. These symptoms continued, with occasional vomiting, for several days; but on Feb. 25

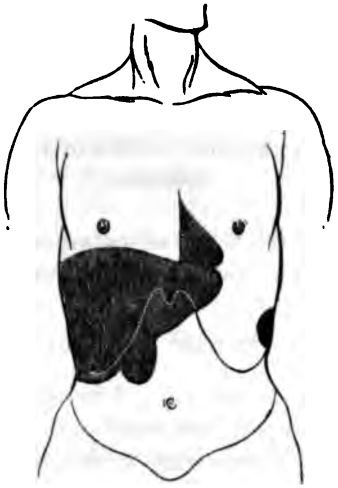

Fig. 16. Shows the Enlargement of Liver and Tumour in case of J. W., on Feb. 20. Compare this with Fig. 3, at p. 4.

temperature had fallen to 99·2°, and on 27th to 97°. On March 1 pulse was down to 72, and tumour in region of gall-bladder had disappeared. On March 4 motions contained much bile, and jaundice was fading. By beginning of April jaundice had almost disappeared, and in May patient was able to resume his employment.

The motions were carefully searched for gall-stones for ten days subsequent to Feb. 24, but none were found. Possibly a gall-stone may have either become disintegrated, or slipped back into gall-bladder. During acute stage the patient was treated with alkalies, ammonia, ether, belladonna, and opium. During convalescence, strychnia appeared to relieve flatulence, and disappearance of jaundice was encouraged by warm baths and diaphoretics.

LECTURE V.

ENLARGEMENTS OF THE LIVER.

SUPPURATIVE INFLAMMATION—PYÆMIC ABSCESSES—TROPICAL ABSCESS.

GENTLEMEN,—The first form of enlargement of the liver to which I desire to draw your attention to-day is that due to

IX. PYÆMIC ABSCESSES.

The abscesses which are often developed in the liver in the course of pyæmia are for the most part many in number and small in size, and in these respects they differ from the tropical abscess, which is usually single and often attains a large size, so as to form a distinct tumour. The clinical characters vary in accordance with this anatomical difference, and with the different conditions under which the hepatic disease occurs. Those of the pyæmic abscess are as follows:—

1. There is enlargement of the liver, usually of moderate extent, but sometimes so great that the lower margin of the organ reaches to the umbilicus.

2. The enlargement is uniform in every direction, and does not produce any bulging of the ribs. In exceptional cases only one of the abscesses enlarges somewhat more than the others and forms a small bulging tumour at the epigastrium; and in cases of still rarer occurrence, the lower margin of the liver, as felt through the abdominal parietes, has a nodulated character from the presence of several small abscesses or inflammatory deposits along its free margin.

3. No fluctuation can be felt in the enlarged liver. The abscesses are rarely large enough to admit of this. Only in those rare cases where one of the abscesses enlarges so as to form a bulging in the epigastrium, or where a small quantity of pus

becomes encysted between the liver and abdominal wall (Case LXX.), is anything approaching to fluctuation perceptible.

4. Pain and tenderness are always present. They are often among the first symptoms noted, and are usually acute in consequence of some of the abscesses being near the surface of the liver, and of the inflammatory action being propagated from them to the superimposed peritoneum. The pain is often increased by coughing or by a long inspiration, so that in consequence the respirations are quick and short, and mainly thoracic.

5. Jaundice is present in the majority of cases—in fully four-fifths; but the possibility of its absence must be kept in view in diagnosis. The intensity of the jaundice varies. In most cases it is due to the morbid condition of the blood to which the term pyæmia is applied, just as jaundice is known to result from other blood-poisons, and then it is usually slight, and the motions are still tinged with bile-pigment; but if the inflammation be due to an ulcer of the biliary passages, excited by the pressure of an impacted gall-stone, the jaundice may be intense and the excrement devoid of bile-pigment.

6. Pyæmic abscesses of the liver rarely interfere with the portal circulation. Accordingly there is no enlargement of the veins of the abdominal parietes, and only in exceptional cases (from implication of a large branch of the vein), ascites. Occasionally fluid is thrown out into the peritoneum as the result of peritonitis. The spleen is usually enlarged, owing, not to obstructed circulation, but to the tendency of that organ to enlarge in consequence of the morbid condition of the blood, as happens in most diseases originating in a blood-poison.

7. The constitutional symptoms are important in diagnosis. They are mainly those of fever, at first hectic and ultimately typhoid in its type. Rigors afford material assistance in diagnosis; but it is well to remember that they are not a necessary symptom. The rigors at first occasionally recur at such regular intervals that the attack simulates ague; errors in diagnosis are constantly committed from this fact not being remembered (Case LXVIII). On the other hand, the possibility of rigors and even pyrexia resulting from the passage of a gall-stone, without any secondary inflammation of the liver, must not be lost sight of. The temperature exhibits great oscillations; sometimes it is normal, at others it reaches 104° or 106°; in rare cases there appears to be no elevation of temperature, perhaps from the fever paroxysms being so short as to escape

detection (Case LXXII.). Profuse perspirations during
are less frequently absent than rigors. Day by day the pa
becomes more emaciated and prostrate, and not unfreque
there is vomiting and attacks of diarrhœa. As the dis
advances, typhoid symptoms such as a dry brown tongue,
lessness, delirium, involuntary evacuations, &c., make
appearance.

8. The course of the disease is rapid, usually ranging
two or three weeks to three months. I have never known
latter limit exceeded, although Leudet mentions a case w
lasted as long as five months.[1] This rapid course may
service in diagnosing cancer, in which the duration is us
more protracted, from pyæmic abscesses of the liver.

9. The diagnosis will also be assisted by keeping in
the circumstances under which the disease usually oc
Among them the following hold a prominent place:—

a. External injuries and surgical operations. When
ptoms like those above described follow either of the causes
mentioned, there need be no difficulty about the diagnosis.
most of the cases, however, which come under the care of
physician depend upon internal causes, and then the diffi
is increased.

b. Ulceration of the stomach or intestine. I have in se
instances known pyæmic abscess of the liver supervene
simple ulcer of the stomach, and I shall relate to you im
diately the particulars of a case where this occurred. It may
follow ulceration of any portion of the intestine, such a
ulcer of the appendix vermiformis, or dysenteric ulcerati
the colon, or even cancerous ulceration of the stomach or b
Pyæmic deposits in the liver, however, only occur in except
cases of intestinal ulceration, probably for the same reason
general pyæmia only occurs in exceptional cases of ext
injury (see p. 178).

c. Ulceration of the gall-bladder or of the bile-ducts
give rise to pyæmic abscesses of the liver, which in this
may be a sequel of gall-stones. I shall narrate to you pres
cases where an ordinary attack of biliary colic came in
to be followed by fatal inflammation of the liver (Case
It may be added that when the common bile-duct is
by a gall-stone or from any other cause, the ducts in th

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[1] Clin. Méd., Paris, 1874, p. 33.

of the liver may become dilated into irregular cavities full of pus,[1] or may rupture and form small abscesses, and in either case there may result many of the symptoms of pyæmic hepatitis.[2]

d. In a former lecture I brought before your notice instances in which a suppurating hydatid cyst appeared to be the starting point of pyæmic abscesses in the liver (see pp. 114 and 119).

e. Lastly, any suppurating ulcer or cavity on or near the outer surface or in the interior of the body, especially if in connection with diseased bone or communicating with the external atmosphere, may induce pyæmia with secondary deposits in the liver. On more than one occasion, for instance, I have found these deposits in the liver resulting from a tubercular vomica in the lungs, ulcerative endocarditis, calculous pyelitis, &c.

When the signs and symptoms already enumerated supervene on those of any of the maladies now referred to, the probability of pyæmic abscesses of the liver ought at once to suggest itself. But occasionally the primary disease is latent, and the first symptoms are those of inflammation of the liver. Even then, however, the probability of pyæmic abscesses ought to suggest itself in English practice, inasmuch as, with extremely rare exceptions, this is the only form of hepatic abscess met with in this country in persons who have never been in a tropical climate.

Treatment.—In pyæmic abscesses of the liver, medical art, it is to be feared, is powerless to avert the fatal result, and can only mitigate the patient's suffering.

1. By hygienic arrangements, by the antiseptic treatment of open sores and wounds, and by evacuating decomposing pus pent up in any part of the body, much can be done in the way of preventing general pyæmia in surgical injuries; but unfortunately in a large number of cases of pyæmic abscesses in the liver that come under the physician, the primary disease is inaccessible.

2. Depletion, both general and local, is contraindicated; but if the pain be very acute it will often be materially relieved by the application of a few leeches to the region of the liver.

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[1] Dr. Legg has recorded an interesting case where in consequence of a gall-stone in the common duct, all the bile ducts became greatly dilated, and an abscess formed in the left lobe of the liver, which found its way into the pericardium and right pleura. Path. Trans. xxv. 133.

[2] It is even said that dilatation of the ducts into suppurating cavities may result from disease of their walls, independently of obstruction. See Dr. Grainger Stewart, Edin. Med. Journ., Jan. 1873, p. 631.

Mustard and linseed poultices are also useful for relieving the pain.

3. Since the discovery of bacteria in the blood of pyæmia much has been written about the internal administration of antiseptics, but proofs are still wanting of their utility. Professor Polli, of Milan, has strongly recommended the sulphites of potash and soda as antidotes for the pyæmic poison. The power which these substances possess of arresting putrefaction or fermentation out of the body it is believed that they can exercise in the living blood. I have tried them repeatedly, and I regret to say that in my practice, in doses of twenty or thirty grains every four hours, they have signally failed. The sulphocarbolates of lime and soda, and the subcutaneous injection of carbolic acid have also been tried, but without any permanently good result.

4. Quinine and mineral acids have appeared to me to be the remedies most generally useful. They support the patient's strength, keep the tongue moist, postpone the paroxysms of pyrexia, and tend to diminish the profuse sweating.

5. The hydrate of chloral, opium, or morphia will be necessary in most cases to relieve pain or procure sleep. If there be much retching, the subcutaneous injection of morphia will be preferable to administering opiates by the mouth.

6. The treatment must often be modified in such a way as to counteract various distressing symptoms which are apt to arise, and more especially vomiting and diarrhœa. For the vomiting, the best remedies are ice, bismuth, hydrocyanic acid, effervescing alkaline draughts, and the application to the epigastrium of sinapisms or of a small blister, followed by the sprinkling of a quarter of a grain of morphia on the blistered surface. For the diarrhœa you must have recourse to vegetable and mineral astringents, and particularly the acetate of lead and morphia, and to opiate enemata and suppositories.

7. The diet must be of as nutritious a character as is compatible with the patient's digestive powers. It ought to consist of such articles as milk, beef tea, and eggs, given frequently, but in small quantities at a time. In most cases it will be necessary to give small quantities of wine or brandy, which ought to be well diluted.

I shall now proceed to relate to you the particulars of a few cases in illustration of the foregoing romarks. In the first case the hepatic disease was the result of an external injury.

CASE LXVII.—*Injury of Cranium, followed by Pyæmia and Multiple Abscesses in Liver.*

Thomas D——, aged 21, was admitted into one of surgical wards of Middlesex Hosp. Aug 16, 1867, with lacerated wounds of scalp, fracture of sixth left rib, and bruise of left shoulder—injuries which he had received from being run over by a cab. He had so far recovered that on Sept. 3 he was able to be out in garden; but on same day he was seized with rigors, followed by febrile symptoms, headache, and loss of appetite. During next two days he had several attacks of severe rigors, like those of ague, followed by moderate perspiration and frequent vomiting.

When he first came under my care, on Sept. 6, he had all the symptoms of blood-poisoning, but without any eruption on skin. Pulse 120; resp. 36; temp. 103°. Alternate fits of chilliness and perspiration. Countenance heavy and depressed; great lassitude; throbbing headache, but mind quite clear; great prostration, and tendency to syncope on sitting up; frequent retching, with tenderness in epigastrium and right hypochondrium. Tongue moist, and but slightly furred. Bowels had been freely opened by medicine. Cardiac and respiratory signs normal. Urine contained a small quantity of albumen, with blood-corpuscles and epithelial casts. A wound in left temporal region was covered with a hard scab, from beneath which about a teaspoonful of dirty, not fetid, pus could be squeezed. Soon after patient's admission he became very restless and delirious; there was no paralysis, but hearing was preternaturally acute. Tongue became dry and brown, and there was frequent vomiting with a tendency to diarrhœa. Tenderness in epigastrium and right hypochondrium continued, and hepatic dulness became much increased, extending down almost to umbilicus; surface smooth. Skin sallow, but no decided jaundice.

Patient was treated mainly according to plan recommended for pyæmia by Professor Polli, of Milan, with large doses of sulphites. Sulphite of soda was given in doses of fifteen grains every four hours. No improvement, however, was observed; and the symptoms above noted continued almost till death, at 9.45 P.M. on Sept. 9.

On examination of body about a square inch of bone, corresponding to wound in scalp, was bare and discoloured. The bone appeared scratched on surface. It was not fractured; but between its under surface and corresponding dura-mater there was about a drachm of pus; veins leading from this to longitudinal sinus contained pale, soft, non-adherent coagula. Liver very large, extending down to umbilicus, and weighing 104 oz. Its tissue was dark and intensely injected, and riddled with innumerable pyæmic deposits breaking down into pus, from size of a pin's-head up to that of a walnut. Spleen large, weighed 10½ oz., and was dark and firm, but contained no infarctions. Both kidneys much enlarged, weighing

95·2°), and pulse was from 84 to 92. A bedsore formed over sacrum, and patient gradually sank and died on Dec. 11.

Autopsy.—Extreme emaciation. Two pints of flaky fluid in peritoneum. Intestines deeply injected and coated with recent lymph, most abundant over cæcum and ascending colon. Mucous membrane of cæcum and colon free from ulceration or cicatrix. Appendix vermiformis unusually long, its upper two inches pervious and healthy, but the distal half thickened, indurated, impervious, and adherent to cæcum. No foreign body or concretion found. Both lobes of liver studded with numerous small circumscribed abscesses, several of them in back part of right lobe merging into one. Glands in fissure of liver enlarged and suppurating. Gall-bladder distended with about two fluid ounces of thin mucous fluid; no calculus, abrasion, or redness of lining membrane of gall-bladder or of ducts. No ulceration of stomach, or of intestine. Granular consolidation of lower lobe of right lung. Other organs healthy.

In the next case a cancerous ulcer of the stomach appeared to be the exciting cause of the disease in the liver. The case has additional interest from the fact that there was a small fluctuating tumour at the epigastrium, caused by a circumscribed collection of pus between the liver and abdominal parietes.

CASE LXX.—*Cancerous Ulcers of Stomach followed by Pyæmic Abscesses of the Liver.*

In June 1867 I was requested by Dr. Rogers, of Dean Street, to see a patient under his care. He was a man, aged 45, whose father and sister were said to have died of cancer. For several months he had been losing flesh, and had suffered pain after food, and other symptoms of indigestion, but not vomiting. About May 19 his symptoms became worse, and he first consulted Dr. Rogers. He then began to suffer from a constant pain in right side, febrile symptoms, dyspnœa, and a frequent dry cough, and on May 23 and again on the 28th he had severe attacks of vomiting. About June 2 a slight swelling was first noticed in epigastrium, and he became slightly jaundiced, and when I saw him on June 8 with Drs. Anstie and Rogers, there was considerable jaundice, with great emaciation and prostration. Pulse quick and feeble, and a tendency to nocturnal perspiration, but no rigors. Tongue moist, clean, and red; no vomiting or diarrhœa, and motions contained bile. Liver much enlarged, and in epigastrium there was a very painful prominent tumour, about size of half an orange, extremely elastic, and indeed apparently fluctuating. An exploratory puncture was made into this tumour, but only a few drops of blood came away. Patient became daily more emaciated and prostrate;

tongue became dry and brown, and jaundice increased although stools still contained bile-pigment. On June 24 he died from exhaustion. Throughout there had been no rigors, and only slight perspiration during sleep.

On examining body, liver was found of almost twice normal size; signs of recent peritonitis over its outer surface; glandular tissue was extremely congested, and was studded with inflammatory (not cancerous) deposits up to size of a walnut, which were pale yellow, granular, and very friable, but which had not yet softened into pus. Between left lobe and abdominal wall there was about an ounce of pus circumscribed by firm adhesions. This accounted for fluctuating tumour felt during life; the fine trocar had probably passed through the abscess into liver, and thus no pus had been obtained by the puncture. On opening stomach an ulcer was found about 2 in. from the pylorus; edges and base of this ulcer were indurated from what microscope showed to be cancerous tissue, and surface of the ulcer was ragged and sloughy.

The next case which I shall refer to is that of a lady 23 years of age, whom I saw in consultation with the late Mr. Young, of Sackville Street, in November and December 1861. It affords an illustration of pyæmic abscesses of the liver supervening on gall-stones.

CASE LXXI.—*Attacks of Biliary Colic followed by Pyæmic Abscesses in Liver.*

On November 30, 1861, I was called to see Mrs. ——, aged 23, who had been married only four or five months. Two years before she had suffered for several weeks from jaundice, with severe attacks of biliary colic. Ten days before I saw her the jaundice had returned, and during same period she had been suffering from severe paroxysms of pain in right hypochondrium, often accompanied by vomiting. Although, notwithstanding patient's age, her history was clearly one of gall-stones, yet, after making allowance for her hysterical temperament, the symptoms led to the suspicion that there was something more. The pulse was 100, and there was an unusual amount of tenderness in region of liver, and particularly in situation of gall-bladder. Hepatic dulness was increased; there was also great increase of splenic dulness. The jaundice was of moderate intensity; and motions, though very pale, were not entirely devoid of bile-pigment. Leeches, followed by warm fomentations, were applied to right hypochondrium, and repeated doses of opium were prescribed.

During first week in December patient had frequent attacks of vomiting, and on 4th she miscarried at third month. After this she became much worse. She had repeated attacks of rigors, lasting for half an hour or more, and often followed by involuntary discharge of light-yellow fluid from bowels. She had also frequent and severe paroxysms of retching, and the pain in right side became so intense that she could not take a long inspiration without crying out. Patient was never free from pain and tenderness in region of duodenum, but the intense pain was decidedly paroxysmal; sometimes, but not always, the paroxysms seemed to be induced by patient moving or taking a long inspiration. Pulse varied from 100 to 120; cheeks flushed, but no perspirations; much thirst, but even fluids were at once rejected from stomach. Jaundice diminished; motions always contained bile, and at last were almost natural in appearance. All treatment failed to give relief; patient became rapidly emaciated, and was occasionally delirious during night; and towards the end tongue was dry and brown, and sordes collected on lips and teeth. Death took place on Dec. 23.

On post-mortem examination liver was found to be large, and entire substance of both lobes studded with an immense number of circumscribed abscesses, varying in size from a pea to a small orange, and filled with yellow flaky pus; outer surface glued by recent lymph to diaphragm and adjoining organs. Hepatic and common ducts pervious and contained bile. Gall-bladder collapsed, its cavity being scarcely larger than a hazel-nut, and its coats much thickened. A gall-stone, somewhat larger than a pea, was found impacted at commencement of cystic duct, and mucous membrane in contact with the concretion was ulcerated, and partly converted into a blackish slough. Beyond this cystic duct was obliterated. Gall-bladder contained about a dozen calculi of smaller size, but no bile; fundus firmly adherent to duodenum, and between these two viscera was a closed cavity containing gall-stones, equalling in size and number those found in gall-bladder itself; corresponding mucous surfaces of duodenum and of gall-bladder marked by an extensive cicatrix. These appearances were probably the result of a direct passage of gall-stones through the fundus of the gall-bladder into the bowel in the attack two years before death. Mucous membrane of first three inches of duodenum intensely injected but not ulcerated; inner surface of the stomach and intestines presented nothing abnormal. Spleen was four times its normal size. In addition to the coating of recent lymph, capsule of liver at several places presented old thickening and firm adhesions. Lungs congested, but otherwise normal.

The following case is interesting from its remarkably latent character, and for the absence of pyrexia. The coexistence of plugging of the femoral vein with enlargement and tenderness of the liver, bile-pigment in urine, and sallowness of the skin suggested that the patient might be suffering from pyæmic inflammation of the liver, and this diagnosis was made, notwithstanding the absence of rigors or perspirations, which are not unfrequently absent in pyæmia arising from internal causes. I was, however, scarcely prepared to meet with pyæmic inflammation of the liver with no elevation of temperature, during at least five successive days, which this case shows to be possible.

Case LXXII.—*Gall-stones causing Ulceration and Perforation of Cystic Duct—Pyæmic Hepatitis and Thrombosis of Femoral Vein—Absence of Pyrexia.*

Mary Ann S——, aged 53, adm. into Middlesex Hosp. April 13, 1869. Never had any symptoms pointing to gall-stones, and excepting diseases of childhood, had enjoyed good health, and been able to work as charwoman till three months before admission, when she began to complain of excruciating pain and swelling in right leg, and lost appetite and flesh. She continued going about, however, till ten days before admission, when she was seized with rather severe pain in epigastrium, nausea, vomiting, thirst, and increased prostration.

On admission patient was of stout habit, very prostrate, dejected and apathetic. She was also very restless and sleepless at night, but she had no headache or delirium and her memory was good. Her chief complaint was uncontrollable vomiting, so that she could retain nothing on her stomach. Much thirst; tongue red and dry all over. Decided tenderness in epigastrium and below right ribs; liver extended 2 in. beyond ribs in right nipple line, where it measured vertically 5 in.; its surface smooth. No ascites or enlargement of spleen; bowels regular. Pulse 84, feeble; physical signs of heart and lungs normal; skin cool; no eruption; temp. 98° Fahr. Urine contained bile-pigment, but no albumen. Face slightly sallow, but conjunctivæ white. Right thigh and leg swollen throughout; and great tenderness along whole course of right femoral vein.

Treatment consisted mainly in giving milk and ice by mouth, and brandy and beef-tea by rectum; but patient continued to sink. On April 16 hiccup set in, with coffee-ground vomiting, and watery motions containing blood. Death occurred on April 18.

Neither before nor subsequently to admission into hospital had patient at any time rigors or perspirations; and during whole time that she was under observation, although temperature was taken twice daily, at no time did it rise above 98° Fahr. in axilla, and only once did it reach $99\frac{3}{4}$° under tongue.

Autopsy.—Two and a half inches of fat in abdominal parietes. Right iliac and femoral veins plugged throughout, coagulum for 3 or 4 in. at brim of pelvis being decolorised and adherent, but below this black and non-adherent. Liver adherent to duodenum and colon, and on removing it gall-bladder was torn across, allowing a number of polyhedral gall-stones about size of plum-stones, and covered with pus, to escape. These gall-stones had been enclosed in a cavity bounded by liver and surrounding parts, with its internal surface ulcerated, and communicating by a large ragged opening with cystic duct. Beyond this, cystic duct was closed by adhesions, but hepatic duct was pervious. No bile in gall-bladder. Liver studded with numerous inflammatory deposits, up to size of a cherry, most of them consisting of a firm, translucent, greyish material, which in some instances was softening into an opaque fluid, like pus. The firmer material was made up of branched fibre-cells; and the yellow fluid of oil-globules and compound granular bodies, but no true pus-corpuscles. The intervening portions of liver, spleen, and kidneys were soft, apparently from rapid decomposition. Heart flabby, its lining membrane stained with blood-pigment. Lungs congested, with small sub-pleural ecchymoses.

Case LXXIII. was remarkable for the large size attained by the liver, and for the absence of any cause of the hepatic inflammation, excepting the softening tubercle in the mediastinal glands.

Case LXXIII.—*Multiple Abscesses of Liver—Softening Tubercle in Mediastinal Glands.*

Ann C——, aged 57, a cook, adm. into Middlesex Hosp. under my care Jan. 13, 1868. Her father and mother had both lived to upwards of 80, and, with exception of an umbilical hernia and a great tendency to vertigo, she herself had always enjoyed good health until her present illness, which commenced a week before Christmas with acute pain in region of liver, stretching round back to left side. This pain was accompanied by febrile symptoms, loss of appetite and sleep, and by a swelling and tightness in upper part of abdomen, which increased daily. On Jan. 5 her face and eyes had been noticed to be slightly yellow.

On admission patient was an extremely corpulent woman, whose skin and conjunctivæ presented a slightly jaundiced tint, and who was so weak as to move her great bulk with difficulty in bed. The abdomen was enormously enlarged, measuring 53 in. at umbilicus. Moderate œdema of both lower extremities, but no distinct thrill of fluid in abdomen, and percussion yielded a clear sound in both flanks. The great size of abdomen appeared due partly to an enormous subcutaneous deposit of fat, and partly to enlargement of liver, which measured 9 in. in right mammary line, and which reached fully 5 in. below margin of right ribs. So far as an examination could be made through the thickened abdominal parietes, enlargement of organ appeared to be uniform in every direction, and its surface was hard and smooth. On pressure over it there was decided tenderness, and a pain shooting from point of pressure to back. Tongue dry and red down centre; much thirst; no vomiting; bowels regular. Pulse 108. Heart's sounds very feeble, but no bellows-murmur. Respirations embarrassed and thoracic; sonorous râles at bases of both lungs. Urine of a dark amber colour, with a copious deposit of lithates; no albumen. Mind clear. Temperature 98° Fahr.

Patient was ordered the day after admission a draught containing a drachm of sulphate of magnesia three times a day, but on Jan. 15, after three doses, bowels were so purged that a mixture of nitro-hydrochloric acid and gentian was substituted. Diarrhœa, however, persisted, motions being watery and dark-brown; tongue continued dry; temperature rose to 101·4°; on nights of Jan. 17 and 18 patient had much low delirium; and in afternoon of Jan. 19 she died suddenly by syncope while attempting to get out of bed.

Autopsy.—Fat in abdominal parietes measured fully 4 in. in thickness. Peritoneum contained about three pints of turbid serum, with small flakes of lymph. Liver enormously enlarged, its lower margin projecting about 5 in. beyond that of right ribs; it weighed 256 ounces, and was studded throughout with innumerable minute abscesses, the projection of which from outer surface gave to this a coarsely granular aspect. The portions of hepatic tissue which remained were in an advanced state of fatty degeneration, but there was scarcely a quarter of an inch of organ free from purulent deposit. Gall-bladder much distended with innumerable black polygonal concretions, from size of a small cherry to that of a grain of sand; majority were small, and resembled grains of coarse gunpowder; the larger ones were found on section to be white internally, and to be composed of cholesterin. Common bile-duct patent, and after careful examination, no ulceration could be discovered in lining membrane of gall-bladder or of any of ducts, nor in mucous membrane of stomach or intestines. No pus in portal vein or embolism of hepatic artery. Spleen large and soft. Kidneys rather large and pale, but appeared normal. Right Fallopian tube dilated into a cyst the size of an orange, containing a dark thin fluid, and with several small vegetations attached to its lining membrane. A fibrous tumour size of a walnut in walls of uterus. At apices of both lungs there were old tubercular cicatrices, but no cavities, and in anterior mediastinum were two or three collections of pus, formed by suppuration of tubercular lymphatic glands. Heart pale, flabby, and friable, and in an advanced stage of fatty degeneration.

X. TROPICAL HEPATITIS AND ABSCESS OF THE LIVER.

The pathology of tropical abscess of the liver has been a subject of much discussion, and one on which opinions are still divided. The frequent coexistence in the tropics of abscess of the liver with dysentery has naturally led pathologists to connect the two lesions, some, like Annesley, maintaining that the dysentery is the result of the hepatitis; others, that the hepatitis is the result of the dysentery; while a third class, like Dr. Abercrombie, have suggested that the frequent concurrence of the two maladies is merely the result of accident. The doctrine now most generally accepted in this country is that propounded nearly thirty years ago by Dr. G. Budd, viz. that the hepatic inflammation is the result of purulent absorption from the ulcerated colon, or in fact that the pathology of tropical abscess is identical with that of the pyæmic abscesses of this country.[1]

Considering how frequently in this country abscess of the liver is secondary to ulcers of the bile-ducts, stomach, or bowels, or other sources of purulent absorption, it would indeed be extraordinary if dysenteric ulceration of the colon never led to a like result, as some have contended. The fact that fatal dysentery with ulceration uncombined with hepatic abscess is a common occurrence in India is no argument against hepatic abscess occasionally resulting from dysentery, any more than that, in Europe, pyæmic abscesses only occur in exceptional cases of intestinal ulceration, or of the other sources of purulent absorption already enumerated. Something more than an open sore is necessary for the formation of pyæmic deposits. The discharges from the sore must be in a peculiar state of decomposition. The causes of this decomposition may be extrinsic or intrinsic, but where there is no such decomposition there is no pyæmia.

But a large number of the abscesses of the liver met with in tropical countries cannot be ascribed to dysentery, or to a pyæmic origin, or to mechanical injury. More than twenty years ago, I stated that this was the result of my observations on the diseases of Burmah,[2] and the facts which have since been published by Morehead,[3] Bristowe,[4] Frerichs,[5] McLean,[6] and others, appear to me to be perfectly conclusive on the matter. These facts are of a fourfold nature.

1. Cases are not uncommon in tropical countries where there has been abscess of the liver, and where the patient has

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[1] Dis. of Liver, 3rd ed. p. 82.

[2] Observ. on the Climate and Diseases of Burmah. Edin. Med. and Surg. Journ. 1854, pp. 245–7.

[3] Researches on Diseases in India, 1856, ii. p. 10.

[4] Path. Trans. 1858, ix. p. 250.

[5] Frerichs, Treatise on Dis. of Liver, Eng. Ed. ii. p. 116.

[6] Article on Suppurative Infl. of Liver, in Ryenolds s System of Med. iii. p. 324.

recovered without any symptoms of dysentery before, during, or after the hepatic malady. I shall give you the particulars of such a case immediately (Case LXXIV.).

2. In many cases where there has been a concurrence of hepatic abscess and dysentery, the symptoms of the former malady have preceded those of the latter. A case of this sort was recorded by me in the eighth volume of the 'Pathological Transactions' (p. 237), and similar cases are referred to by Morehead, Waring, and Bristowe.[1]

It may perchance be argued that in the cases included under these two heads dysenteric ulceration was really present, but that its symptoms were latent. Dr. Dickinson, for instance, has recorded a case where extensive dysenteric ulceration and a large abscess of the liver were found after death without any symptoms during life to lead to a suspicion of either malady.[2] But although such an explanation may apply in a few exceptional cases, it is obviously inapplicable to such results as those obtained by Mr. Waring, who states that of 300 cases of hepatic abscess proving fatal in India, in only 82 cases, or in 27·3 per cent., was the hepatitis preceded by symptoms of dysentery.[3]

3. The most conclusive cases, however, are those in which the patient has died of hepatic abscess, and no sign of dysenteric ulceration has been found after death. I shall give you immediately the details of a case of this sort, in which, it is important to add, there had been a considerable amount of diarrhœa during life (Case LXXIV.). Morehead observed 21 fatal cases of abscess of the liver 'without any sign of intestinal ulceration,'[4] while in 204 cases of abscess of the liver collected by Waring there were no ulcerations, cicatrices, or abrasions in 51, or in exactly one-fourth.[5] Lastly, in the Pathological Museum at Netley there are 48 specimens of tropical abscess of the liver, of which in 34 the abscess was uncomplicated with any intestinal lesion.[6]

It is clear, therefore, that although dysenteric ulceration of the bowel may occasionally lead to pyæmic deposits in the liver similar to those met with in this country, many cases of tropical abscess are independent of such an origin. Few Indian physicians would, I think, admit the validity of Dr. Moxon's argument that intestinal ulceration or cicatrices would be found in all cases of tropical abscess of the liver if the bowels were examined with sufficient care.[1]

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[1] Dr. James Finlayson has pointed out how hepatic abscess may lead to congestion and even ulceration of the colon. Glasgow Med. Journ., Feb. 1873.

[2] Path. Trans. 1862, vol. xiii. p. 120.

[3] An Enquiry into the Statistics and Pathology of Abscess of the Liver. Trevandrum, 1854.

[4] Op. cit. ii. p. 12.

[5] Op. cit.

[6] McLean, op. cit. iv. 324.

4. It appears to me that the etiology of hepatic abscess receives further elucidation from an anatomical point of view. The abscesses of the liver which are met with in this country, and which are the result of absorption from an open sore, are usually, if not always, small but numerous. On the other hand, in most cases where abscess of the liver is met with in the tropics, there is but one abscess which attains to large dimensions, or in exceptional cases there may be two or three. In a case recently under my care 160 ounces of pus were drawn off by tapping during life. Abscesses of the liver, answering to this description, are almost unknown in this climate or in temperate climates generally, except in persons who have sustained some local injury of the liver, or who have at one time resided in the tropics—an extraordinary fact, if their cause be the same as that of multiple abscesses. Even when dysentery occurs in temperate climates no such abscesses are found in connection with it. Out of many hundreds of cases of dysentery which occurred in Millbank Prison during thirty years, we are told, on the authority of the late Dr. Baly, that not one was complicated with hepatic abscess.[2] In Germany, it is stated by Heubner, that hepatic abscesses are sometimes met with after dysentery; 'they are, however, multiple and of embolic source,' and it is added that these abscesses ought not to be confounded with the ordinary abscess of the tropics.[3] These facts alone are sufficient to show that tropical abscess of the liver is independent of dysentery. To argue that the large size and singleness

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[1] Path. Trans., 1873, xxiv. 116.

[2] 'When the close relation subsisting between dysentery and suppurative disease of the liver is considered, it cannot but appear remarkable that amongst the many hundreds of cases of dysentery which have occurred in the Millbank Prison during the last seven years, not one has been complicated with hepatic abscess. The medical records of the establishment, too, which reach back to the year 1824, afford no grounds for even a suspicion that such cases ever occurred amongst the prisoners.' Yet, 'in this dysentery in the Millbank Prison the disease of the mucous surface, both as to its seat and in its nature, has been the same as in the dysentery of India, with which hepatic abscess is so frequently associated.' Gulstonian Lectures on Dysentery, 1847.

[3] On Dysentery. Ziemssen's Cyclop. of Med., Amer. Ed. 1875, i. 546, 556. Rokitansky also, in his dissections of cases of dysentery, never found the liver visibly diseased; while in France, Broussais, who reported 17 fatal cases of dysentery with dissections, does not mention his having found abscess of the liver in any one instance, although the condition of this organ is frequently mentioned. Baly, op. cit.

of the tropical abscess is due to the longer time it lasts as compared with the swift course of the multiple abscesses met with in pyæmia[1] is, it appears to me, to disregard the clinical history of the two maladies; in the tropics one enormous abscess may form in a fortnight, but both in tropical and temperate climates small multiple abscesses may be found after an illness which has lasted for months.

For these reasons I have proposed to designate the single large abscess so common in warm climates the *Tropical* Abscess, to distinguish it from the *Pyæmic* Abscess which is the common form in this country.

In suggesting these designations it is not contended that small multiple abscesses of the liver are unknown in the tropics;[2] but, so far as I have been able to ascertain, this form is never met with except in connection with dysentery, or with some other source of purulent absorption. It must not be forgotten that a single large abscess, like a suppurating hydatid in the liver, may be a source of infection and of small secondary abscesses. The single tropical abscess may also coexist with dysentery, but from the large number of cases in which both dysentery and hepatic abscess are independent of each other, it follows that when they coexist, they are either the effects of a common cause, which in certain persons will produce either of the diseases separately, or of a concurrence of causes which individually will cause only one of the diseases. The latter view is favoured by the fact that a single large abscess is not found in connection with dysentery in temperate climates. Supposing, for example, what is probably the truth, that dysentery is the result of a poison inhaled, or swallowed in drinking water, and that hepatitis may be caused by a chill in a person whose liver has been congested by a residence in a hot climate, aided by intemperance, irritating ingesta, and exposure to the malaria of tropical fevers (see p. 134), it is readily conceivable that in a country like India where these causes so often operate simultaneously, attacks of dysentery and hepatitis—combined as well as separate—should not be uncommon.

The distinction drawn above between pyæmic and tropical abscess is far from being one merely of pathological curiosity; it has a most important bearing both on prognosis and treatment. The pyæmic abscess is much the more serious and fatal malady of the two; the danger is from the blood-poison and not from the local disease; and recovery from it rarely, if ever, occurs. The tropical abscess again is a local malady, not unfrequently recovered from; the abscess may discharge itself through the lung, the stomach, or the bowel, or externally, and thus the patient may recover—terminations not met with in the case of pyæmic abscesses; and lastly, one of these natural modes of termination of the tropical abscess is advantageously imitated by the surgeon, when he evacuates the abscess by an external opening—a procedure which would obviously be worse than useless in the pyæmic abscess. It follows, therefore, that it is of some practical importance to be able to distinguish during life between the pyæmic and the tropical abscess. The characters of the former have been already detailed; those of the latter remain to be considered. They are as follows:—

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[1] Moxon, loc. cit.

[2] Of 300 cases of abscess of the liver in India collected by Waring, the number of abscesses was not stated in 12; of the remaining 288, there was one abscess in 177; two abscesses in 33; three in 11; four in 17; five to ten in 10; more than ten in 40. An Enquiry into the Statistics and Pathology of some Points connected with Abscess of the Liver, as met with in the East Indies. Trevandrum, 1854, p. 125.

A. In the early stage of the disease, the main clinical features are those of hepatic congestion already described (see p. 131). There is chilliness followed by pyrexia, often of a remittent type, accompanied by pain and tenderness, or oftener by a feeling of weight, fulness, or uneasiness in the region of the liver, and occasionally by pain in the right shoulder, defective respiratory movement of the right ribs, dry cough, a uniformly augmented area of hepatic dulness, and slight jaundice. The enlargement, however, is on the whole less, and the jaundice much rarer than in the congestion of the liver resulting from disease of the heart or lungs. This is due to the circumstance that the branches of the hepatic and portal veins, which are gorged in the latter case, are much larger than those of the hepatic artery, which are the main seat of the congestion that precedes the formation of abscess. But in not a few cases there are no local signs of mischief in the liver, and in fact the only symptoms may be those of an intermitting or remitting fever which may be thought to be malarious.

B. When the inflammation goes on to suppuration, which unless it terminate previously by resolution, often occurs at the end of a week or twelve days, the characters are as follows:—

1. There is enlargement of the liver, which is no longer uni-

form. The natural outline of the area of hepatic dulness is altered, and will bulge upwards, downwards, forwards, or outwards, according to the direction which the abscess takes in each case (see fig. 17, page 193). Not unfrequently there is a bulging of the ribs, with obliteration of the intercostal spaces, or there is a prominence in the epigastrium, or in the right hypochondrium, such as occurs in hydatid tumours.

2. This bulging or tumour is tense, rounded, smooth, and free from any inequalities. In the advanced stage, however, of exceptional cases the margin of the enlarged liver may be nodulated from the development in it of small secondary pyæmic abscesses.

3. Fluctuation can usually be detected in the tumour, which will be more or less distinct according to the distance of the abscess from the surface. The feeling of vibration, however, which can often be appreciated on tapping with the finger over a hydatid tumour (page 56), cannot be elicited in an abscess, owing to the greater thickness of its contents. Another distinctive character of abscess is, that the fluctuation is usually surrounded by a mass of inflammatory hardness.

But although tropical abscess is a common cause of enlargement of the liver, an abscess of considerable size, if deeply seated, may cause no apparent fluctuation, bulging, or even enlargement. This is a fact which I cannot too strongly impress upon your memories.

4. Pain and tenderness are very often absent. Pain, when present, is dull and heavy, and not of that acute character, in the first instance, at all events, so common in the pyæmic abscess. This is because the abscess is usually at first in the interior of the liver. The pain only becomes acute like that of pleurisy, and the tenderness great, when the matter approaches the surface of the liver and excites peri-hepatitis, or stretches the integuments. Thus it is that acute pain often marks the last stage, instead of the commencement, of the morbid process. Some cases are remarkably latent, as far as pain is concerned, throughout their whole course; while in others pain is only produced when the patient takes a long breath, and at the same time pressure is made below the margin of the ribs, or over the lower end of the sternum. A sympathetic pain in the right shoulder is not uncommon, especially when the abscess is situated on the convex surface of the right lobe; but in most cases it is absent. The presence of pain in the shoulder will

undoubtedly increase, although its absence will not diminish, the importance of other symptoms.

5. Ascites, œdema of the lower extremities, enlargement of the superficial veins of the abdomen, and hæmorrhoids are not distinguishing characters of tropical abscess, any more than of hydatid of the liver. Their occurrence in rare cases is accidental, and due to compression by the tumour of the trunk of the portal vein or of the inferior vena cava. Occasionally, fluid is thrown out into the peritoneum as the result of peritonitis.

6. Enlargement of the spleen is rarely present in tropical abscess.

7. Jaundice is a much rarer symptom in the tropical than in the pyæmic abscess. Its occurrence, in fact, if we except a slight icteric tint during the primary stage of congestion, is quite exceptional. Morehead has noted it in only five out of upwards of 120 cases.[1] When it occurs, it has mostly a mechanical origin, and is due to the concurrence of catarrh of the bile-ducts, or to the direct compression of the large ducts by the abscess.

8. The constitutional symptoms are important, as serving to distinguish the tropical abscess from hydatid tumour, and also from the fact that in the absence of local signs the diagnosis must be founded upon them alone. After the occurrence of suppuration, they are mainly progressive emaciation and fever of the hectic type. The frequency of the pulse may be little, if at all, increased; but (unless the abscess has become encysted and quiescent) there is almost always an elevation of temperature to several degrees at some period of the twenty-four hours. Rigors and night-sweats are less prominent symptoms than in the pyæmic abscess. The tongue becomes covered with a grey or yellowish coat, and in the advanced stage it may be preternaturally red and dry and coated with aphthæ. Loss of appetite is a common, but far from invariable, symptom. Obstinate vomiting is present in many cases,[2] and ought always to excite suspicion of abscess of the liver in a tropical climate; it is very apt to occur when the abscess is about to discharge into the stomach or duodenum, and the exhaustion which it entails may be the immediate cause of death. Diarrhœa, or even dysentery, occurs in some cases. The urine is loaded with

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[1] Res. on Dis. in India, 2nd. ed. 1860, p. 373.

[2] See Dr. W. C. Maclean and Sir Joseph Fayrer, Brit. Med. Journ. 1874, ii. 138, 401.

lithates or lithic acid, and contains much pigment; the urea is greatly increased, but when the hepatic tissue has been in great measure destroyed, it may be deficient. Temporary albuminuria, often considerable, is not uncommon. Very often there is a short dry cough, and the respirations are quickened, especially when the abscess is about to perforate the diaphragm, and then also friction may be heard at the base of the right lung.

It is important to remember, however, that a tropical abscess of the liver may be so latent as to reveal itself by neither local signs nor constitutional symptoms. Not unfrequently the only symptoms are debility and paroxysms of fever, which are believed to be malarious, but the real nature of which is first suspected by their failing to yield to large doses of quinine. In some cases, as we shall presently find, even pyrexia may be absent.

9. The duration of tropical abscess of the liver is a matter of some importance in diagnosis. Although it may terminate fatally, or may discharge in some direction within three weeks of the commencement of the symptoms, yet, on the whole, the course of the disease is less rapid than that of the pyæmic abscess. Very often it extends over two, three, or even six months; and cases are not uncommon where a small tropical abscess with thick organised walls has existed for months, or even years, in a quiescent form, and has then undergone enlargement and burst. Some of the cases met with in this country, where a large abscess forms in the livers of persons years after their return from India admit of this explanation; while others are perhaps what Sir James Paget[1] would call 'residual abscesses,' or abscesses formed in or about the residues of former inflammation.

10. The circumstances under which 'tropical abscess' occurs may sometimes be of material assistance in diagnosis:—

a. Its frequency in certain parts of the tropics, and particularly in India and China, and its extreme rarity in temperate climates, except in persons who have visited tropical countries. In the West Indies, curiously enough, it is comparatively rare.

b. It is chiefly met with between the ages of 20 and 45.

c. It is most common in persons of indolent habits, and who have been excessive eaters or intemperate in the use of alcohol. Of 40 cases in which the habits were noted by Waring, 67·5 per cent. were intemperate.

d. The concurrence of dysentery.

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[1] On Residual Abscesses. St. Barth. Hosp. Rep. 1869, v. 73.

11. The diseases most likely to be confounded with tropical abscess are hydatid tumour, inflammatory enlargement of the gall-bladder, pyæmic abscesses, and abscess of the abdominal parietes (see p. 15).

a. A hydatid of the liver is the enlargement most likely to be mistaken for abscess. In both there may be a local projection from the general contour of the liver, presenting fluctuation and occasionally causing bulging of the ribs or a semiglobular tumour in the epigastrium. Tropical abscess is mainly to be distinguished from hydatid by the presence of pain, by its much more rapid course, by its constitutional symptoms, and by the circumstances under which it occurs. The possibility, however, already referred to, of a hydatid tumour suppurating or becoming converted into an abscess must not be lost sight of. An error in diagnosis from this cause is all the more likely to arise if the patient, as often happens, has been ignorant of the existence of the hydatid tumour prior to the occurrence of the acute symptoms due to its taking on inflammatory action. Any doubt will usually be removed by an exploratory puncture, while the treatment in both cases will be the same.

b. The circumstances under which enlargement of the gall-bladder may simulate hepatic abscess and its distinguishing characters will be considered in a subsequent lecture. It may be here observed, however, that a large abscess connected with the liver in a person who has never left this country is in most cases either a suppurating hydatid or an inflamed gall-bladder.

c. The constitutional symptoms of tropical and pyæmic abscesses may be identical. For distinguishing them, we must rely mainly on the form of the enlargement, the circumstances under which each occurs (see pp. 166 and 185), and the greater tendency in the pyæmic abscess to jaundice and symptoms of blood-poisoning.

Treatment.—A. *Before suppuration.* Until a comparatively recent date the two remedial measures mainly relied on in the treatment of tropical abscess of the liver were general bloodletting and mercury. With regard to bloodletting there can be no doubt that in the case of plethoric Europeans but recently arrived in the tropics, in whom the disease often sets in acutely, with full firm pulse and high temperature, it often relieved pain and reduced the fever, but there is no evidence that it prevented suppuration and some suspicion that it hastened it, while all its good effects will be obtained by the application of a few leeches over the liver or round the anus. The same may be said of mercury. Pushed to salivation, it is more likely to favour suppuration than to prevent it, and it will certainly increase the tendency to malarial cachexia and anæmia. Except as an occasional aperient, it ought never to be given. The rules for the treatment of the early stage of tropical hepatitis are the same as those which I laid down in my last lecture for congestion of the liver (p. 134), the remedies on which reliance is mainly to be placed being the chloride of ammonium and ipecacuanha in large doses. The liability to diarrhœa or dysentery makes it necessary to be more cautious in the use of purgatives than in the ordinary hepatic congestions of this country.

B. *After suppuration.* In tropical abscess of the liver, not only may we hope to prevent suppuration by appropriate treatment, but even after it has occurred the case is far from being, as in the pyæmic abscess, necessarily fatal. The treatment, however, for the stage antecedent to suppuration is no longer suitable.

1. Warm fomentations and poultices are still to be applied to the region of the liver; and in the event of acute pain supervening, although this usually indicates an advanced stage of the disease, a few leeches will often give relief.

2. The patient's strength must be supported by mineral acids and vegetable tonics, and in particular by the sulphuric or nitric acid with quinine.

3. Opium is in most cases necessary to relieve pain, to procure sleep, or to allay the harassing cough.

4. Purgatives are no longer called for. If the bowels be constipated, a mild laxative may be given from time to time, but more commonly there is diarrhœa or dysentery, necessitating the use of vegetable and mineral astringents, with opiate enemata or suppositories.

5. The diet must be of a more generous nature than that which is permissible in the stage of congestion; and when the circulation is weak, small quantities of wine or brandy will be necessary.

6. In multiple abscesses, which must be regarded as a local manifestation of a general disease, it is clear that no advantage is to be derived from operative interference, but when there is a single large abscess, the general symptoms being here the result of the local disease, the propriety of evacuating the pus may fairly be entertained. It is no doubt true that a large

abscess of the liver may become encysted and shrivel up, and in this way undergo what may be called a spontaneous cure, independently of rupture, but this is an event so rare that it cannot be calculated on. Recovery also takes place occasionally in consequence of the abscess emptying itself through a bronchial tube, into the bowel, or externally through the abdominal parietes; but the process is tedious, and even when it occurs many patients die of exhaustion from fever, pneumonia, or diarrhœa, to say nothing of their liability to destruction at any moment from the abscess bursting into the pericardium, the pleura, or the peritoneum. In a large proportion of cases, however, the patient dies while the abscess is still confined to the liver.[1] Under these circumstances the expediency of hastening the evacuation of the matter naturally suggests itself.

You will find nevertheless that professional opinion is divided on this important question. Dr. Budd, in his standard work on 'Diseases of the Liver,' considers the dangers of operating so many and so great, that it is better to let matters alone and allow the abscess to open of itself.[2] Some authorities, again, such as Frerichs[3] and Morehead,[4] advocate opening the abscess in selected cases; while others, like Dr. Murray, formerly Inspector-General of Hospitals in Bengal, Dr. Cameron,[5] and Sir Ranald Martin,[6] maintain that 'when we have just grounds for believing that abscess of the liver exists, we ought not to lose a day in evacuating it by puncture, and that we are both justified and safe in endeavouring to hit upon it with a trocar when deep-seated, avoiding the gall-bladder and large veins.'[7] Dr. Cameron, in fact, goes so far as to recommend exploring the liver with a trocar in cases where the existence of an abscess is suspected, though not certain, and has published cases where no pus was found, and yet the patient's symptoms subsided, instead of being aggravated, subsequently to the exploration. Amid such conflicting opinions we may be aided in forming a just judgment by considering, on the one hand, the dangers of the operation, and, on the other, the dangers of non-interference.

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[1] Of 300 fatal cases of hepatic abscess collected by Mr. Waring, the abscess, at the time of death, had not extended beyond the boundaries of the liver in 169; in 48 it had been opened by operation; in 42 it had opened spontaneously into the right lung or thoracic cavity; in 15, into the peritoneum; in 8, into the stomach or colon; in 3, into the hepatic vein, &c. Op. cit.

[2] Op. cit. 3rd ed. 1857, p. 124.

[3] Dis. of Liver, Syd. Soc. Ed. ii. p. 147.

[4] Res. on Dis. in India, 2nd ed. 1860, p. 410.

[5] Lancet, June 6 and 13, Aug. 8, 1863.

[6] Lancet, Aug. 20 and 27, 1864.

[7] Cameron. Lancet, June 6, 1863, p. 631.

The main objections raised against the operation are as follows:—

a. That pus is apt to escape into the peritoneum and excite fatal peritonitis. In most cases, however, when the abscess is near the surface, there would be adhesions which would prevent the entrance of pus into the peritoneum. Morehead speaks of the absence of adhesions as quite exceptional (in only 3 of 76 fatal cases). Moreover, if desirable, it is always possible to produce adhesions.

b. That air will enter the abscess and excite fresh inflammation, or pyæmia. This is an undoubted source of danger; but it is as likely to be incurred if the abscess opens spontaneously into the bowel, a bronchial tube, or externally. Moreover, it may be in a great measure prevented by the use of antiseptics.

c. That the mechanical injury of the puncture is apt to produce hæmorrhage and fresh inflammation in the hepatic tissue. So far as I have been able to ascertain, this is an objection founded on theoretical considerations rather than on actual observation. I have had several opportunities of confirming Dr. Cameron's statement to the effect that a fine trocar can be plunged into the liver without any ill result, and without, in fact, any trace of the puncture being discernible when death occurs shortly afterwards (see Case LXXXII.).

d. That the fatal event may be hastened by gangrene of the tissues around the wound spreading inwards to the liver.[1] This accident has been chiefly observed when the opening has been made in an intercostal space, and then, as Morehead has shown, it occurs alike when a spontaneous rupture takes place, and when a puncture is made.[2] The gangrene is most probably connected with the caries or necrosis of the ribs, which is almost always present in these cases, and which would probably not occur were the abscess opened before the ribs became implicated. This danger might also be averted by opening from below the ribs and by the use of antiseptics.

The chief dangers of non-interference are these:—

a. The abscess daily becomes larger, more and more of the hepatic tissue is destroyed, and ultimately the gland may be

[1] Maclean. Lancet, July 18, 1863.

[2] Op. cit. p. 410.

reduced to a mere sac containing pus, while adjacent organs are compressed and the ribs are eroded.

b. The patient may die suddenly from the abscess bursting into the pericardium, the peritoneum, or the pleura. Not long ago I saw a patient with abscess of the liver beginning to point at the epigastrium. I advised paracentesis, but as the case was not considered urgent this was delayed. Two days afterwards the gentleman died quite suddenly. The abscess, in process of opening into the colon, had leaked into the peritoneum.

c. The majority of patients with abscess of the liver die of exhaustion from hectic fever or diarrhœa, either while the abscess is still confined to the liver, or after it has burst.

Statistics have been appealed to with the object of proving the uselessness of operative interference. Of 81 cases where the abscess was opened, collected by Mr. Waring, only 15 (or 18·5 per cent.) recovered, and of 24 cases recorded by Morehead, only 8, or one-third, recovered. But in many of these cases death was due, not to the operation, but probably to this having been too long delayed, or to proper precautions not having been adopted; while several of Waring's cases were examples of multiple abscesses, for which an operation was obviously unsuited. Moreover, of 203 cases collected by Rouis, where the abscess was not opened, 162 (or 80 per cent.) died.[1]

After duly balancing, then, the dangers of operation against the dangers of expectancy, I do not hesitate to recommend to you the propriety of evacuating the pus, with proper precautions, in a large number of cases of tropical abscess of the liver. The operation may not be free from danger, but to wait in these cases upon Nature, as it is called, is to wait upon Death, and I would suggest for your guidance the following rules:—

a. In all cases where there is a visible fluctuating tumour, operate at once.

b. In cases where the symptoms of abscess of the liver are present, with a distinct tumour projecting from the normal contour of the liver, or causing bulging of the ribs, though there be no perceptible fluctuation, it will be well to operate.

c. When symptoms of abscess coexist with uniform enlargement of the liver, but with no distinct tumour or bulging, if there be any local œdema, or obliteration of an intercostal space, or pain localised to one spot when pressure is made on it, or when the patient takes a full inspiration, it will be well to operate.

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[1] Frerichs, op. cit. ii. p. 136.

d. Where there are no local signs of abscess, but where the constitutional symptoms leave little doubt of its existence, and are severe, one or more exploratory punctures with the aspirator will be advisable (see Case LXXXII.). Even if the abscess be not reached, the direct abstraction of a small quantity of blood from the liver sometimes gives great relief.

e. When from the presence of jaundice or other symptoms there is reason to fear that there are numerous abscesses, it will be better to abstain from any operation.

When the operation is resolved on, it may be performed as follows—

a. When there is distinct pointing with an inflammatory blush of the skin, and the abscess is small, an opening may be made with a bistoury.

b. Under other circumstances a small trocar will be preferable, and it ought to be introduced wherever there is the slightest fulness, superficial œdema, or tenderness.

c. When the abscess is small, not holding more than ten or twelve ounces, it may be completely evacuated, and a drainage-tube fastened in for some days. On the removal of the tube, a tent of lint dipped in carbolic oil may be substituted.

d. In all cases where an external opening is made, the antiseptic measures recommended by Professor Lister ought to be rigidly enforced, and when the abscess is very large, recovery will sometimes be hastened by making a counter-opening and passing a drainage-tube through both openings.

e. When the abscess is very large, it will be better to evacuate it by instalments at short intervals, carefully excluding the air on each occasion. For this purpose Bowditch's syringe or Dieulafoy's aspirator are well adapted.[1]

f. In the exceptional cases where no adhesions exist, it will be prudent to produce them by the local application of caustic potash before puncturing, but when the puncture is made in an intercostal space this proceeding is unnecessary.

The first of the following cases is an excellent illustration of tropical abscess of the liver independent of dysentery, notwithstanding that after the formation of pus diarrhœa was a prominent symptom. It is a matter of regret that the abscess was not punctured; but twenty-three years ago this operation was rarely practised.

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[1] The reader is referred to an interesting case recorded by Professor Maclean, C.B., where recovery followed the removal of 108 ounces of pus by the aspirator. Lancet, 1873, ii. 39.

CASE LXXIV.—*Tropical Abscess of Liver—No Dysenteric Ulceration of Bowel.*

Private H. C——, aged 33, of the 2nd European Bengal Fusileers, was admitted under my care into the Military Hospital at Prome, on Nov. 12, 1853. His habits had been very dissipated; he had suffered from many attacks of fever and congestion of liver, and shortly before his admission he had been exposed almost continuously for three weeks to wet on the decks of steamers, during passage from Calcutta to Rangoon and from Rangoon up the Irrawaddi to Prome. He had never had dysentery. He began to suffer from fever and pain in right side in first week of October during passage from Calcutta, but his condition did not prevent him attending to his duty until a few days before admission, when pain in side became much more severe.

On admission, pulse 112; skin hot. Much pain in region of liver, and stretching up to right shoulder; pain was greatly increased by coughing or taking a long breath, and there was considerable tenderness on pressure over epigastrium and below right ribs. Hepatic dulness in r. m. l. measured 6 inches. Posteriorly and upwards margins of hepatic dulness were normal, and the increased size appeared due to a bulging from the lower margin. No fluctuation and no jaundice or ascites, but there was less movement of ribs in respiration on right side than on left, and frequent cough. Tongue moist and coated white; frequent vomiting; a day or two before admission bowels had been relaxed, but at time of admission they were costive. Some scalding in micturition; urine was high-coloured; sp. gr. 1027; it contained no albumen, but deposited crystals of lithic acid.

Patient was cupped to 8 oz. over liver, and during first week after admission was treated with calomel and opium, and subsequently with nitro-muriatic acid, quinine, opiates, and wine.

On Nov. 18 diarrhœa came on, with profuse night-sweats, but no rigors. Vomiting continued, and tongue was clean, very red, and deeply fissured. Cough and scalding in micturition had abated, but vomiting and diarrhœa persisted, notwithstanding use of remedies. On Nov. 20 tongue dry and brown. Patient became very emaciated, but was comparatively free from pain until Nov. 26, when he was seized with acute pain, shooting up from region of liver to right shoulder. On following day this had subsided; and after this there was but little vomiting or purging, and symptoms were mainly those of hectic fever, with increasing prostration, until Dec. 1, when there was noticed below right ribs, rather to right of mammary line, a distinct smooth rounded bulging, with obscure fluctuation in centre. Hepatic dulness in right mammary line was now 8 in., increase being due to a projection downwards from lower margin of normal area of hepatic dulness. There was also considerable bulging of lower right ribs. Patient was now free from pain; vomiting and purging had ceased; but cheeks were sunken and presented a hectic flush, fever and night-sweats continued, tongue was dry and brown, and teeth coated with sordes. On Dec. 8 patient was in a state of extreme prostration; on following day mind was wandering, and at 9.30 P.M. he died.

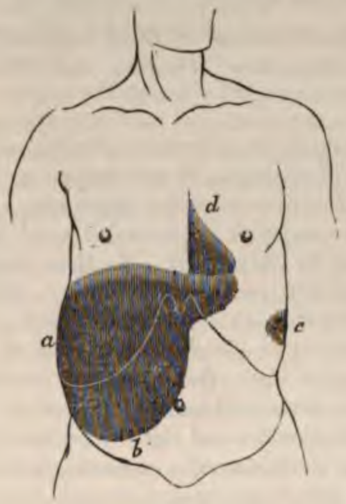

Fig. 17 represents the outline of Hepatic Dulness, and the bulging of the ribs (*a*) in H. C.——, on December 2, 1853.

On examination of body ten hours after death, one enormous abscess was found in right lobe of liver. It contained upwards of four quarts of pus, having a reddish tint, and composed of pus-corpuscles, with oil-globules and hepatic cells undergoing disintegration. Walls of abscess were formed by ragged masses of hepatic tissue coated with inflammatory products; at two places walls were very thin, one situated below margin of right ribs, and corresponding to tumour observed during life, and the other posteriorly near mesial fissure. Stomach and intestines presented no trace of cicatrices or of recent ulceration. Spleen, lungs, and heart normal. Old adhesions between apposed surfaces of left pleura; and cavities of heart, but particularly right, contained large masses of decolorised fibrin.

Case LXXV. is another example of tropical abscess independent of dysentery. Death was due to the supervention of pyæmia and secondary abscesses upon the opening. The good effects which immediately followed the operation make one regret that the matter was not drawn off by instalments with a fine trocar, and that more thoroughly antiseptic measures were not adopted.

CASE LXXV.—*Tropical Abscess of Liver independent of Dysentery—Free Opening—Secondary Abscesses and Diarrhœa—Death by Exhaustion.*

Foogeek Kitche, aged 39, a Japanese juggler, adm. into Middlesex Hosp. Jan. 5, 1871. Left Japan 16 months before and went to Madras, where he remained a month and drank much gin. Never had dysentery and had always enjoyed good health until three months after reaching this country in July 1870. He then began to suffer from weakness, loss of appetite, perspirations during sleep, and occasional pain in right side, and since then he had lost flesh. One month before admission he was obliged to give up work, and at this time he first noticed a swelling below right ribs, which had increased considerably.

On admission was emaciated and complained of pain and swelling in right side. In epigastrium and right hypochondrium was a tumour projecting from liver about size of a cocoanut, causing slight eversion of lower right ribs, dull on percussion, smooth, very elastic but yielding no vibration, and but slightly tender even on free manipulation. Hepatic dulness in r. m. l., including tumour, 5½ in.; posteriorly liver did not rise above normal level. Tongue moist and furred; moderate appetite, and no retching; considerable thirst; no jaundice; bowels regular. Pulse 80; heart and lungs normal. Temp. 101¼°; still perspires during sleep. Urine 1030, loaded with lithates, but free from albumen. Was ordered quinine and nitric acid.

Jan. 9.—Is weaker, and has had more pain in tumour, which is larger. Has required morphia to make him sleep. No rigors, but still perspires profusely at night. Pulse has varied from 68 to 84; and temp. from 99° in morning to 103° at night. After a preliminary exploratory puncture a large trocar was introduced into tumour, and 30 oz. of thick pus of a brick-red colour let out. Cavity was washed out with a solution of chloride of zinc (gr. x ad ℥j), and cannula was left in and covered with lint soaked in carbolic oil and with carded oakum.

Jan. 13.—Operation was followed by great relief. Patient slept without morphia, had no night-sweats, and temperature became normal in evening as well as morning. Last night, however, sweating returned, and to-day pulse 96 and temp. 100·3°; very little discharge. On withdrawing cannula 10 oz. of pus, not fetid, escaped. Cavity was again washed out with a solution of chloride of zinc, and a piece of elastic tube was substituted for cannula.

Jan. 20.—During last week patient, though weak, has felt much better. There has been very free discharge from opening; pulse has varied from 60 to 84, and temp. has never been above normal. Appetite has been excellent, and for last three days patient has had meat, eggs, and porter. To-day, for first time, discharge has been slightly offensive, and patient does not feel quite so well. Porter was discontinued, and cavity was ordered to be washed out daily with a weak solution of carbolic acid.

Jan. 27.—Is weaker, but appetite has continued good; has had no rigor, very little perspiration, and free discharge, not fetid, from wound. Temp. has been normal, except for a few hours on *Jan.* 21, when it rose to 101°. On *Jan.* 23 he had four loose stools, and since *Jan.* 24 there has been a troublesome cough. Yesterday evening, on commencing to syringe out cavity, patient was seized with severe pain in hepatic region and profuse perspiration, which lasted an hour, Only a very little reddish matter came away, but this morning discharge is free and deeply tinged with bile. Pulse 64; temp. 96·8°. Indiarubber tube was withdrawn, and a piece of lint soaked in carbolic oil inserted in wound instead.

Feb. 1.—Very little discharge has come away from wound, and on *Jan.* 29 little that came was fetid, but to-day discharge is more copious. Patient has occasionally complained of rather severe pain in region of liver, and night-sweats have returned. No rigors, and temp. has always been normal until to-day, when it is 101°. Bowels are relaxed, and patient is getting much thinner.

Patient now became rapidly weaker. Night-sweats and diarrhœa were uncontrolled by treatment. Temp. varied from 99° to 102·8°, but at no time were rigors noted, and pulse was usually 120; urine free from albumen. Death took place on *Feb.* 4.

Autopsy.—Right lobe of liver was glued by soft adhesions to abdominal parietes over a space about 3½ in. in diameter, and was more firmly connected to diaphragm above and behind where abscess came close to surface; but greater part of surface of liver presented no sign of inflammation, old or recent. Sac of abscess had evidently contracted, for it would not have held more than 16 oz. of fluid. Its walls were very thick, and composed of an external firm fibrous layer several lines in thickness, and of an internal lining of softer honeycombed material. In remaining portion of liver were several abscesses from size of cherry to that of walnut, with no thickened walls and evidently quite recent. Base of right lung was glued to diaphragm by soft lymph, and right pleura contained about an ounce of flaky fluid. Spleen healthy; kidneys congested; large intestine presented no trace of cicatrices, thickening, or other sign of old dysentery; but in cæcum and ascending colon mucous membrane was intensely injected, and rugæ were plastered with large flakes of granular exudation, two small patches of which were also found in ilium just above valve.

In Case LXXVI. the abscess discharged itself through the right lung, and the patient made a rapid recovery. It is a good illustration of the most favourable direction that the abscess can take.

CASE LXXVI.—*Abscess of Liver discharging through right Lung—Recovery.*

On May 2, 1874, I was consulted by Dr. L., about 35. Eight weeks before he had returned from Gold Coast, where he had suffered from fever and pain in right side. On reaching this country he had felt quite well, and had gone about. Three weeks after arrival he had got a rigor one night after going to theatre, and ever since he had been laid up with fever. Temperature had been as high as 105°. At first there had been considerable delirium, and latterly profuse night-sweats, but no return of rigors. At my visit great prostration; pulse 108; temp. 102°; resp. 48. Hepatic dulness reached up to right nipple; it did not come too low, but there was distinct bulging of right lower costal cartilages. Frequent dry cough; crepitation at base of both lungs; and sharp stitch in right side. Tongue thickly coated with yellow fur; bowels confined, and had been so throughout; urine dark, and loaded with lithates.

Ordered ammon. chlor. gr. xx, pot. bicarb. gr. xv, and tinct. op. ♏ v, every six hours; colocynth and calomel pills. Quin. sulph. gr. x while perspiring. Milk, beef-tea, and claret.

On morning of May 6 he suddenly began to cough up pus of a reddish-brown colour and mawkish smell, and in next 24 hours brought up about two pints. Breath had odour similar to that of pus. A mixture of quinine and mineral acids was now substituted for chloride of ammonium &c.

Almost immediately after bursting of abscess into lung there was a fall in pulse, temperature, and respirations; night-sweats diminished and general condition improved. Expectoration of pus continued for about ten days. On May 11 temp. rose for a few hours to 104°, but after this it was normal; and on June 2 patient had so far recovered that he was able to go to Scotland. He had no relapse, and 16 months afterwards (Sept. 25, 1875) he was well and stout, and on examination, no trace of former illness could be discovered, except slight impairment of breath sound at base of right lung. Oct. 1876; still well.

In Case LXXVII. the abscess also burst upwards through the diaphragm, but the result was fatal.

CASE LXXVII.—*Abscess of Liver opening upwards through Diaphragm—Secondary Abscess of Lung.*

I show you here a specimen which I removed some years ago from the body of a patient—a man, aged 34, who died in this (Middlesex) hospital—and which illustrates the bursting of a large abscess upwards through the diaphragm. In this case patient had suffered some years before from dysentery in India and Malta. His symptoms during the nine days that he was in hospital before his death were hectic fever and emaciation, dyspnœa, cough, and purulent expectoration, with a painful enlargement of liver producing an outward bulging of ribs. Hepatic dulness extended only 2 in. below margin of ribs in r. m. l., but upwards it reached to third intercostal space. The enlargement felt smooth, but did not involve whole organ uniformly. Tongue unusually red; no vomiting, jaundice, or diarrhœa, but abdomen generally tender, and distinct evidence of fluid in peritoneum.

After death, three or four pints of flaky serum were found in peritoneum. Liver firmly adherent to diaphragm and abdominal parietes, and in upper part of right lobe was an abscess as large as a cocoanut, which had perforated diaphragm so as to be bounded above by base of right lung. The abscess was enclosed in a dense capsule of areolar tissue, and contained yellow pus with large fibrinous flakes. In lower lobe of right lung was another abscess, the size of a large orange, distinct from former, and containing pinkish pus. Descending colon and sigmoid flexure much contracted; their coats thickened; mucous membrane slate-coloured, but presented no recent ulcers or distinct cicatrices.

In the two following cases the abscess opened into the bowel—probably the colon; but there was a subsequent history of refilling of the cavity and discharge on repeated occasions. I do not remember having seen this occurrence referred to by authors, but from my experience I am inclined to think that it is a not uncommon sequel of the bursting of a large hepatic abscess into the bowel. Of nine cases where the abscess seemed to open into the bowel, and of which I have notes, there was a similar history in six; and in a tenth case the abscess opened first into the bowel and then into the lung.

CASE LXXVIII.—*Abscess of Liver opening into Bowel—Frequent refilling of Cavity with Pus. Death by Diarrhœa and Exhaustion.*

On May 25, 1871, I was consulted by Mr. K., aged 50, on his return from Ceylon, where he had been for 25 years. He had been suffering for nine months from bilious diarrhœa and occasional vomiting. He had considerable pain about the liver, which was somewhat enlarged and tender, and with this there was loss of appetite, pyrexia, night-sweats, occasional rigors, prostration, and emaciation. After this I saw him from time to time until his death in February 1875. At first he improved much under treatment, but on April 9, 1872, after several weeks of great agony an abscess burst, and he discharged at one sitting three quarts of pus from the bowels, and almost a like quantity on twelve different occasions during the next fortnight. After this he was never well. He had constant diarrhœa. But now and then, three or four times in course of the year, diarrhœa would stop for 10 or 14 days, and then he would have rigors, pyrexia, enlargement and pain of liver, all of these symptoms ceasing with a free discharge of pus from bowels. On one occasion after rigors and pyrexia he vomited several ounces of pus and blood. The tongue and mouth at last became red and aphthous, so that he could take little food, and death from exhaustion ended his sufferings. There was no post-mortem examination.

CASE LXXIX.—*Tropical Abscess of Liver opening into Bowel—Frequent Relapses—Superficial Pointing—Free Incision—Recovery.*

Mr. N——, aged 44, was sent to me on April 7, 1873, by Dr. R. J. Black, of Canonbury. He had returned on 14th of previous November from China and Japan, where he had not been very temperate. On voyage home, when at Aden, he had been first seized with symptoms of acute congestion of liver, but he was not obliged to take to bed until Dec. 20, about which time a painful swelling appeared below right ribs, and he suffered from rigors and night-sweats. Swelling continued to increase until beginning of February, when he discharged a large quantity of pus by bowel. Swelling subsided, and general symptoms improved. When I saw him he was thin and weak, but he was free from pain and fever, and liver was not enlarged.

Three or four days after seeing me he was again seized with rigors and pyrexia, and there was pain in hepatic region and some bulging of right costal cartilages and epigastrium; and when I saw him again on April 22, although more acute symptoms had subsided, bulging persisted, and he perspired much during sleep. After this he got much better, swelling again subsided, and for a fortnight he was able to go to business in City; but on May 24 he was once more seized with rigors and pyrexia, followed by a return of painful swelling in same situation as before. Swelling increased and became soft and fluctuating in centre, while patient had profuse night-sweats and grew daily thinner and weaker.

On June 25 a free incision was made by Mr. De Morgan into swelling, and nearly two pints of thick pus of a brick-red colour let out. Cavity was washed out with a strong solution of chloride of zinc (gr. x ad ℥j), and a piece of elastic tube was introduced into cavity and secured; and through this, cavity was washed out daily with a weaker solution of chloride of zinc (gr. iij ad ℥j): end of tube was covered with carded oakum. On June 30 tube was removed and wound dressed with lint and carbolic oil. The operation gave immediate relief; fever and pain at once ceased; and within a week night-sweats had also ceased, appetite was good, and patient was beginning to gain flesh and strength. He had several relapses of fever after this, but ultimately he made a good recovery, and in January 1875 he was in enjoyment of good health and attending to business.

Case LXXX. is an illustration of a large hepatic abscess in a person who had never been out of England, although there were doubts at the time whether the abscess had originated in the liver or in the areolar tissue about the kidney. There was ulceration of the colon, but this appeared from the history and post-mortem appearances to be secondary to the abscess of the liver. The specimen was exhibited by me to the Pathological Society, and the case is recorded in the eighth volume of the 'Transactions.'

CASE LXXX.—*Large Abscess of Liver opening into Ascending Colon.*

J. P——, a man aged 40, was admitted into St. Mary's Hospital, under care of Dr. Sibson, on April 18, 1856.

He stated that he had always enjoyed good health and that, although he had been in habit of drinking a good deal of malt liquor, he had never been addicted to spirits, and had, on the whole, been a temperate man. He had never been abroad. About a month before admission he 'took cold,' and was seized with a shooting pain in right hypochondriac region, which on second day became so extreme as to prevent his working. He went to bed, where he remained until day of admission, pain in right side continuing without intermission except when relieved by opium.

After admission, there was found to be a great fulness in right hypochondriac and lumbar regions, with a feeling of a resisting mass extending downwards as far as crest of ilium, and forwards to within 3 in. of linea alba. This space was universally dull on percussion, and dulness was continuous with that of liver; upper margin of hepatic dulness was not elevated and dimensions of left lobe appeared normal; swelling was of a doughy consistence, and presented indistinct fluctuation. Tongue loaded; bowels rather confined. Urine voided three or four times a day, and acid; sp. gr. 1020. Pulse 108, weak.

Poultices of linseed meal were applied over swelling, while iodide of potassium (gr. ij ter die), gentle laxatives, opiates, and stimulants were prescribed internally.

On April 24 he had an attack of erysipelas of face which continued for four or five days. On April 26, during this erysipelas, he was seized with violent diarrhœa. This ceased in a great measure after four or five days, and he then felt himself greatly better; appetite had improved, pain had gone, swelling and dulness were much diminished, and calls to make water less frequent. He continued to improve until May 11, on which day he had a return of severe pain and diarrhœa, with purulent stools. Pain was referred chiefly to a spot about two inches below margin of ribs, in a line with right nipple. Stools were of a light buff colour and very offensive. This diarrhœa resisted all treatment, and soon patient's strength began to give way. He had febrile exacerbations towards evening, and profuse perspirations during night. Pulse varied from 100 to 125, and was very weak; tongue became dry and brown; and he gradually sank until death at 10 P.M. on May 27. Four days before death swelling in right side was observed to have greatly diminished, dulness in right lumbar region not extending farther forward than a perpendicular line drawn from middle of crest of ilium to ribs.

Post-mortem examination forty-one hours after death.—On opening abdomen, extensive adhesions of viscera and other indications of peritonitis, entirely limited to right side, peritoneum on left side being normal. These adhesions of viscera on right side rendered their examination extremely difficult; whole of anterior margin of right lobe of liver firmly adherent to peritoneal surface of abdominal wall, while under surface of anterior edge, along with gall-bladder, was in intimate union with transverse colon. Texture of liver was pale. In lower part of right lobe was an abscess as large as two fists, containing a quantity of fluid fæculent matter of a light yellow colour. This abscess involved almost whole of that portion of lobe to right of fissure of gall-bladder, and extended to within half an inch of its upper surface; upper two-thirds of walls of abscess formed by hepatic tissue, rough and ragged without any limiting membrane; lower part was completed by kidney, anterior layer of fascia lumborum, and about 3 in. of ascending and transverse colon. This portion of colon communicated freely with cavity of abscess. Its upper wall next abscess presented a cribriform appearance, all that remained of it being a few narrow bridles, passing transversely and easily torn across. Extensive ulceration of adjacent portion of ascending colon, and slight ulceration of Peyer's patches in ileum. Kidneys anæmic, spleen soft and friable. Thoracic organs healthy, left cavities of heart containing blood, right being empty.

The next case which I shall mention is a good illustration of the benefit which may often be derived from evacuation of the abscess.

CASE LXXXI.—*Tropical Abscess of Liver—Puncture with a large Trocar—Recovery.*

Mr. C. D——, aged 23, consulted me on June 11, 1867. He had arrived from Calcutta the day before, and gave the following account of himself. He had resided in Calcutta for about three years, and had lived freely, but had never suffered from dysentery. He had been taken ill about end of March with fever and rapidly increasing prostration. He had no pain in side, no diarrhœa, and no jaundice, but about April 12 a tumour made its appearance below right ribs, which rapidly increased until 19th, when it was opened with a large trocar and upwards of a pint of matter let out. The cannula was left in the wound, and on 21st the patient was put on board the overland steamer in so prostrate a state that he was hardly expected to recover. He slowly improved, however, during voyage, and cannula was removed at Aden about a fortnight afterwards. I found an opening with pouting granulations about half-way between umbilicus and ribs, and 2 in. to right of mesial line, from which about two drachms of thin pus escaped daily. The patient was weak and anæmic, but in other respects appeared to have nothing amiss. He was treated with mineral acids, quinine, and iron, and within three months he had regained his usual health and strength. There was then no evidence of enlargement of liver, and the opening had permanently closed. (With the exception of an attack of gout in January, which he had previously suffered from, and of which disease his father had died, he remained in good health until he returned to India in February 1868.)

Case LXXXII. is an example of tropical abscess of the liver without local signs, and it shows also that the liver may be freely probed for pus with a fine instrument without any harm resulting.

CASE LXXXII.—*Deep-seated Abscess of Liver—Exploratory Punctures without result—Pleuro-pneumonia—Death.*

Mr. C. B——, aged 42, consulted me on March 22, 1873. He had just returned from India, where he had resided for nine years, and where he had enjoyed fair health until 3rd of last January, when he had been seized with dengue fever, followed by an attack of acute dysentery which had quite ceased by Jan. 25. Ever since, however, he had suffered from weakness, uneasiness in right hypochondrium, slight perspirations during sleep, cold creeping sensations down back, and audible pulsation in right ear on lying down, which kept him awake at night. He had also a feeling of heaviness at epigastrium after meals; bowels rather costive; urine dark, and loaded with lithates; pulse 108. Right lobe of liver slightly enlarged, measuring 5 in. in r. m. l. Ordered a scruple of chloride of ammonium three times a day; occasional aperient of blue pill and rhubarb; to rub red iodide of mercury over liver; and to avoid stimulants. On March 31 he returned a good deal better and complaining chiefly of atonic dyspepsia, for which he was ordered nitro-muriatic acid with quinine and pepsine. After this he got very much better and married. On May 8 he returned, and stated that five days before (during cold east winds) he had been seized with 'ague;' severe rigors every afternoon followed by great heat and profuse sweating, and that ever since he had lost appetite. Urine was again loaded with lithates and contained a trace of albumen; bowels confined. Ordered an aperient of calomel and rhubarb, an effervescing alkaline draught, and gr. xv of quinine during sweating stage. These large doses of quinine, followed first by gr. v, and subsequently by gr. x, three times a day, failed to arrest the paroxysms of fever, which recurred once or oftener every day, and at irregular hours. Careful observation showed that he was never entirely free from pyrexia, pulse varying from 84 to 108, and temperature from 100° to 102°. Urine was still loaded with lithates and contained a trace of albumen; there was profuse sweating during sleep, and patient became daily thinner and weaker. Although there was no local bulging, œdema, or tenderness over liver, an abscess in that organ appeared to offer the only solution of the symptoms, and it was determined to explore for it. Accordingly, on May 27 a small trocar was introduced by Mr. De Morgan to depth of three inches into right side of epigastrium where liver appeared to be slightly enlarged, and on June 12 two other punctures to depth of 4 or 5 in. were made, one in front a little above first, and the other at back between eighth and ninth ribs, and the aspirator was applied. On both occasions only a few drops of blood escaped. The punctures were not followed by any pain or aggravation of general symptoms, but patient became daily weaker. On June 20 signs of pleuropneumonia of lower lobe of right lung set in; after this rigors and perspirations ceased, hepatic dulness in r. m. l. receded one inch from nipple, and prostration rapidly increased until death, which was preceded by slight hæmoptysis on June 28.

Autopsy, by Dr. H. W. Hubbard, who had attended patient since May 12.—An abscess containing about 10 ounces of thick yellow pus in upper and back part of right lobe of liver, within half an inch of surface. Rest of liver congested, but otherwise healthy. Not the slightest sign of peritonitis or extravasation in situation of punctures, nor in fact anything to show where they had been made. Upper surface of liver corresponding to abscess, and also base of right lung, adherent to diaphragm by recent lymph. Extensive pneumonia of lower lobe of right lung. The abscess was still confined to liver, and diminution of hepatic dulness in front, observed during life, was due to liver having bulged less forward, and pointed more up towards lung.

My main object in drawing your attention to the following case is that an absence of any elevation of temperature led, in the first instance, to an error in diagnosis. Notwithstanding the previous history of dysentery and diarrhœa, and the evidence of gastric and hepatic derangement immediately before the acute attack, the severity and the paroxysmal character of the pain, associated with tenderness and obscure swelling in the region of the gall-bladder, but unattended by any sign of fever, pointed to a calculus in the cystic duct as the probable cause of the attack. The progress of the case, however, made it probable that the cause of the pain was an abscess, which ultimately discharged itself into the bowel. The precise seat of the abscess is somewhat doubtful. The fact of the pain being at first referred to the lower part of the abdomen, and the almost instantaneous discharge of matter per rectum after the sensation of bursting, suggested that it might be in the neighbourhood of the rectum; whereas the previous history of dysentery, the symptoms of hepatic derangement immediately before the acute attack, and the circumstance of there being an obscure swelling with tenderness in the region of the liver, which disappeared after the discharge of matter, were in favour of a hepatic abscess. The supposition of an abscess in the gall-bladder, secondary to obstruction of the cystic duct by a gall-stone, was rendered improbable by the fact of the abscess discharging itself, without any evidence of antecedent obstruction of the common duct (jaundice), although it is possible that an inflamed gall-bladder distended with pus might have discharged itself into the colon. But whatever was the seat of the abscess, the point of interest was the same, viz. that an abdominal abscess existed, which for days caused intense pain, but none of the usual constitutional symptoms of pyrexia. The cessation of the symptoms in the second attack, without any obvious discharge of matter, was probably due to the discharge being less sudden, and to the pus being obscured by fæcal matter.

CASE LXXXIII.—*Hepatic (?) Abscess, without Elevation of Temperature.*

On Feb. 18, 1875, I was called to see Mr. A——, aged 40, in consultation with Dr. Collyer, of Enfield. Mr. A—— had been in China for a good many years, but for last five years he had resided in London, or in neighbourhood. About 1865 he had contracted dysentery in China, and ever since he had suffered from chronic dysenteric diarrhœa, from three to five stools daily, often containing blood and mucus, and sometimes attended by tenesmus. Still his appetite had kept good, and his body was fairly nourished. Nine weeks before I saw him he began to complain of loss of appetite, nausea, and occasional vomiting, and he became sallow, his bowels acting as usual. He went about, however, until five days before my visit, when he was seized with severe pain in abdomen, which gradually increased until night of Feb. 17, when he was rolling about in agony for several hours, and was only relieved after repeated and large doses of opium. The pain at first had been referred rather to lower part of abdomen; but when I saw patient it was restricted to region of gall-bladder, where a distinct, but not well-defined, prominence, about size of an orange, could be felt. With the pain there had been frequent rigors and retching, but repeated observations with thermometer failed to discover any elevation of temperature, and there were no perspirations. At time of my visit, temp. 99·5° F.; pulse 76; tongue thickly coated, yellowish; much flatulent distension of abdomen.

Patient was ordered rest, hot poultices to abdomen, and an effervescing soda draught, with liq. op. sed. ♏ xv, every four hours while pain continued severe.

Pain was but little relieved by treatment. It continued intense until evening of 21st, when, while sitting in an easy-chair before fire, he experienced a sensation as if something had given way in region of gall-bladder, and immediately a large quantity—about a pint—of yellow matter was discharged per rectum. This was examined microscopically both by Dr. Collyer and by his partner, and was found to consist of true pus. A good deal of matter came away next day, and a smaller quantity on 23rd, but from moment of first discharge pain ceased. On Feb. 24 patient passed only a little blood and mucus. After this stools became more natural; appetite returned; swelling and tenderness disappeared; and patient went on well until March 8, when pain returned in severe paroxysms as before, and again no elevation of temperature. On March 13, when I paid a second visit to patient, pulse 68; temp. 99·2°; still paroxysms of intense pain and retching; tenderness and obscure swelling in region of gall-bladder. Pain persisted for five or six days longer, and then subsided—this time without any obvious discharge of matter. On April 1 patient visited me in town, and then complained of nothing except his usual diarrhœa, which by April 14 had greatly abated under the use of creasote and opium.

Whatever was the seat of the abscess in Case LXXXIII., there can be no doubt that in the following case an enormous abscess existed in the liver, and was rapidly increasing in size, without any elevation of temperature. The case in this respect was no doubt exceptional, but I am inclined to think that it is far from unique. At the very time at which this patient was under notice I saw another in whom a similar observation had been made. It is difficult to account for the absence of pyrexia while extensive suppuration is going on in these cases, except on the supposition that the morbid process here entails the destruction of an organ which contributes in part to the maintenance of the animal heat; and yet, so far as we know, the temperature is elevated in most large abscesses of the liver.

But, whatever be the explanation, the possibility of a large abscess existing in the liver without any elevation of temperature is a fact of great clinical importance. The case was further interesting as showing the origin of the secondary abscesses in inflammation of the small branches of the portal vein.

CASE LXXXIV.—*Tropical Abscess of Liver—History extending over 3½ years—Absence of Pyrexia—Paracentesis—Death.*

Lieut. M., aged 25, consulted me on Dec. 1, 1873. In Nov. 1868 he first went to India. During 1869–70 he had ague repeatedly, and in 1871–72 he had repeated attacks of congestion of liver, but it was not until Dec. 1872, after much exposure to sun, that he first began to get seriously out of health. He then got enlargement with pain in liver, loss of appetite, retching, and obstinate constipation, jaundice, and sleeplessness, and after some weeks attacks of shivering followed by sweating. After three months the more acute symptoms subsided, but he remained very weak, and the left lobe of liver was still large and painful. In April 1873 he left India on sick leave, and in June he arrived in England. He continued to improve until middle of November, when he was seized with pain in right lobe of liver, increased by lying on left side, loss of appetite, and great prostration, and when I saw him liver measured 6 in. in r. m. l., but no sign of fluctuation, retching, or diarrhœa. Pulse 108; great pallor. He was ordered saline aperients and large doses of chloride of ammonium, and occasional doses of blue pill. Under this treatment he speedily improved, and I did not see him again; but he had an attack of a similar nature in his liver about once a month. In one of these attacks he kept his bed for six weeks, had profuse sweating every night, and lost nearly 2 stone in weight. After attack was over he quickly rallied, and could go through much hard work, but with the return of the attack he was at once prostrated. During attacks liver always enlarged, and urine became very dark and turbid, but in intervals it was pale and clear. He had no rigors with attacks. In Aug. 1875 he returned to India, but attacks continued to recur; in intervals he had sometimes diarrhœa, and he was again sent home on sick leave in March 1876. On April 27 I saw him for the second time in one of the attacks. He was very prostrate; great pallor; liver large—6 in. in r. m. l.; decided tenderness over right floating ribs at back, but no bulging or fluctuation. Temp. normal. No jaundice. Four days before he had been so well that he had felt none the worse for walking fifteen miles; and two days afterwards (April 29) he appeared again perfectly well, and liver was reduced to 5 in., its upper border in front still ascending about an inch too high. On May 12 he had another attack; much pain in liver, which again became enlarged; great thirst; obstinate constipation; urine loaded with brick-dust sediment: pulse 100; sleeplessness, but no elevation of temperature; no night-sweats, rigors, or retching. After about a fortnight a fluctuating swelling began to appear in right loin, which rapidly increased. Patient became wasted and worn; slept none, owing to great pain; tongue dry and red; and on May 30 diarrhœa set in, five or six stools a day; but all this time temp. never rose above normal, even under tongue; no rigors, and scarcely any sweating.

On June 6 he was admitted into St. Thomas's Hospital. Very prostrate. P. 122. T. 98·4° to 99·2°. Resp. 30. Hepatic dulness in front extended from nipple to one inch below ribs = 7 in.; lower margin very tender. In right loin was a prominent fluctuating swelling measuring 8 in. by 9, very tender. Girth of abdomen over this swelling 19½ in. on right side, 15¾ on left. Much pain and restlessness. T. coated and red; bowels loose. Urine turbid, and contained albumen.

On June 7, 18 ounces of thick reddish-brown pus were drawn off from abscess by aspirator. No relief followed.

June 8.—P. 124; T. 98·4° to 98·6°. Tongue dry, red, and glazed: five or six loose motions; 17 oz. of similar pus drawn off by exhausting syringe.

June 10.—No improvement. P. 124 to 150. Temp. 98·2° to 99·5°. An incision was made into swelling, and a pint of pus let out; cavity was washed out with a solution of chloride of zinc (xx gr. to 1 oz). and a drainage-tube fastened in, and wound dressed antiseptically.

This operation gave great relief, but diarrhœa persisted. On June 12 he began to have frequent retching; on June 13 constant hiccough; on June 16 rapid breathing; tongue dry and glazed, and latterly aphthous, total loss of appetite; albumen and lithates in urine, and daily increasing prostration until death on June 19. After operation temp. never exceeded 99·6° and varied between this and 97·5°. The principal remedies employed were large doses of quinine, opium, bismuth, and stimulants.

Autopsy.—No recent peritonitis, but general adhesions on lower surface of liver and over upper surface of right lobe. Lower margin of right lobe of liver did not extend beyond margin of ribs in front; but posteriorly, projecting from its under surface, was a large abscess cavity, extending upwards to lower border of seventh rib, and downwards to crest of ilium, lying in front of right kidney, and bounded in front by a thin layer of peritoneal adhesions. Immediately adjoining this, but more in substance of liver, was an irregular abscess about 1½ in. in diameter, communicating on the one hand by a fistulous passage with the large abscess cavity, and on other by a narrow sloughy opening (apparently recent) with neck of gall-bladder. This abscess contained several small gall-stones, and in gall-bladder were about a dozen black gall-stones, size of peas, and some thin pus. Throughout liver were several smaller abscesses, one near anterior end of falciform ligament, which had almost burst and was covered by a layer of recent lymph; another, size of an apple, near upper surface of left lobe; and on under surface of left lobe, near anterior margin, a rounded depressed cicatrix. On cutting into liver also were a number of sharply defined pale yellowish patches, from 6 to 18 lines in diameter, surrounded in some instances, but not in all, by a narrow congested zone. Lobules in pale patches were defined, though here and there outline confused; appearance seemed due to local anæmia, but some of patches were softened in centre. On microscopic examination, Dr. Greenfield ascertained that all the vessels in the pale areæ were filled with coagula. The branches of the portal vein were filled with adherent coagula, and their coats thickened and infiltrated with leucocytes, which were also accumulated around them. The branches of the hepatic artery exhibited the reaction of amyloid degeneration. The hepatic cells were swollen and full of granular or fatty matter; many appeared to be breaking up. The hepatic vessels were filled with coagulum, but coats were not thickened. Liver weighed 111 oz. Numerous minute round and oval cicatrices of former ulcers throughout colon and rectum, and also in lower part of ilium; walls of bowel not thickened. Some glands in fissure of liver, large and softened in centre into pus. Kidneys slightly enlarged, soft, and flabby; cortices swollen; amyloid reaction of Malpighian tufts. Spleen 11 oz., firm, with early waxy degeneration. Patches of recent bronchial pneumonia in lower lobe of both lungs; heart healthy.

LECTURE VI.

ENLARGEMENTS OF THE LIVER.

CANCER.

GENTLEMEN,—The next form of enlargement of the liver, the clinical characters and treatment of which have to be considered, is that which is due to cancerous deposit.

XI. CANCER OF THE LIVER.

Cancer of the liver may be recognised by the following clinical characters.

1. The size of the liver is increased, and not uncommonly the enlargement is very great, so that the organ fills a great part of the abdominal cavity. A cancerous liver has been known to weigh 384 ounces, or about seven times the normal weight.[1] The enlargement is progressive, and in the softer forms of cancer may be so rapid that a weekly increase may be noted. On the other hand, it must be remembered that the liver may contain a considerable amount of cancer, and yet the enlargement may not be appreciable during life. The liver may have been originally a small one, and the addition of the cancer may not cause it to project beyond the costal arch, or the lower margin may be overlapped by a distended bowel. You will remember the case of Mary T——, a very fat woman, 54 years of age, who died recently in the hospital, of apoplexy supervening upon white softening of the brain (with hemiplegia), and whose liver was unexpectedly found to be studded with large cancerous nodules, although the organ did not project beyond the costal arch, and there had been no symptoms during life of disease of the liver. A similar observation was made in two other cases, which I shall detail to you (Cases XCV. and XCVI.). I have known a cancerous liver weigh only 27 ounces.

2. The enlargement is usually irregular, from the presence

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[1] See Budd, Dis. of Liv. 3rd ed. p. 407, and Path. Trans. xviii. p. 145.

of nodular excrescences of cancer projecting from the surface or from the margin of the liver, which can often be felt on palpation, and are sometimes even visible through the abdominal parietes. Occasionally the cancerous deposit forms one large excrescence or tumour at a particular part of the organ. Dr. Bright has recorded some remarkable cases in which the tumour was confined to the left lobe, and projected downwards into the abdomen, or upwards into the left side of the chest;[1] and the specimen I show you here, obtained from the body of a patient who died under my care in the Fever Hospital, is another illustration of the same condition (Case XCIV.). More commonly a number of nodular outgrowths, about the size of cherries or small oranges, project from the portion of liver which is opposed to the abdominal parietes (see fig. 18). Care must be taken not to mistake for such excrescences the rigid bellies of the recti muscles (see page 15). It is necessary also to remember that a nodular character is not essential, as might be inferred from some descriptions, to cancerous enlargement of the liver. In certain cases the cancer is not deposited in the liver in isolated nodules, but is infiltrated through the hepatic tissue in such a way that, although the organ may be greatly enlarged, its natural outline is but little altered; and even in

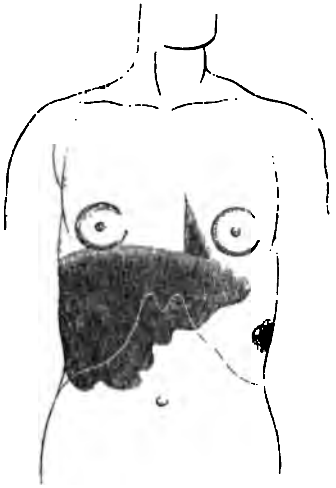

Fig. 18. Shows area of Hepatic Dulness, in Hannah C—— (Case LXXXV.), with nodulated lower margin.

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[1] *Abdom. Tumours*, Syd. Soc. Ed. pp. 261 and 308.

the nodular form of cancer, the portion of liver below the ribs is sometimes quite smooth (Cases LXXXVI. and LXXXVIII.).

3. The enlargement feels very hard and resisting on palpation, and nowhere exhibits any fluctuation. In rare cases some of the cancerous nodules may be so softened as to present obscure fluctuation. Now and then the excrescences develope and grow while the patient is under observation. This circumstance, or the feeling of a depression in the centre of the excrescences, will place their cancerous nature beyond a doubt.

4. A cancerous liver is very often painful and tender on pressure, and the pain radiates to the shoulder, back, and loins. At first there may only be a feeling of weight and uneasiness in the right hypochondrium, but after a time there are paroxysms of lancinating pain awaking the patient at night, and acute tenderness; and both are particularly severe in cases where the growth is rapid, or where, as often happens, there is inflammation of the superimposed peritoneum. The tenderness is usually most marked over the prominent nodules. But many cases of cancerous liver are met with in which there is little or no pain from first to last. Not long ago I had three patients under my care at one time in which this was the case.

5. Jaundice is present in a large number of cases, and when once developed it rarely disappears, The coexistence of enlargement of the liver with persistent jaundice ought always to raise the suspicion of cancer. The jaundice is in rare cases independent of obstruction of the bile-duct; far oftener it results from the compression or obliteration of the bile-duct by a cancerous mass in the liver or by enlarged glands in the portal fissure. If the ducts be not thus compressed, almost the whole of the secreting tissue may be destroyed without any jaundice resulting. Of 91 cases of cancer of the liver collected by Frerichs, 52 died without ever having been jaundiced.

6. Fluid in the peritoneum is observed in more than one-half of the cases of cancer of the liver before the fatal result. Most commonly it concurs with jaundice, or each symptom may exist independently (see Cases LXXXV., LXXXVI., and LXXXVII.). The fluid when copious is usually a simple dropsical collection, due to compression or obstruction with cancerous matter of the trunk or large branches of the portal vein, but the amount is usually small as compared with what is observed in cirrhosis, although now and then it collects with a rapidity rarely seen in cirrhosis. Considering how often the trunk or branches of the portal vein become obstructed with cancerous matter, it is

remarkable that the branches of the hepatic vein usually escape. Very often small collections of fluid are the result of a chronic peritonitis originating on the surface of the liver, and I have known a collection of this sort become encysted above the liver so as to embarrass the diagnosis. Now and then, as in Cases XCIII. and XCIV., blood is thrown out into the peritoneum from a rupture in a fungating or softened cancerous mass in the liver.

7. The superficial abdominal veins are only enlarged in those comparatively rare cases where the portal circulation is seriously obstructed.

8. Enlargement of the spleen is rare, and this constitutes an important distinction of the cancerous from the waxy or cirrhotic liver.

9. The constitutional symptoms, in the first place, are mainly those of deranged digestion, such as nausea, flatulence, and constipation, and occasionally attacks of vomiting or diarrhœa, with aching pains in the muscles and joints and progressive emaciation. A short dry cough is not uncommon. When the cancer grows rapidly, there may be a certain amount of pyrexia (Case XCI.). The urine is invariably scanty and high-coloured, and deposits abundance of lithates and dark pigment unless the patient has been exhausted by vomiting or diarrhœa. Before the disease has lasted long, the patient presents in a marked degree the phenomena of the cancerous cachexia—extreme anæmia, with an earthy chlorotic colour of the integuments (unless there be jaundice), and rapidly increasing debility and emaciation. These symptoms are always aggravated by the coexistence of cancer of the stomach. As a rule, constitutional symptoms precede for some time both pain and jaundice, and for a long time they may be the only evidence of the disease, there being no enlargement of the liver, pain, jaundice, or ascites. A temporary gain in weight and strength under treatment is, however, not incompatible with cancer of the liver.

10. Cancer of the liver is, in most cases (fully three-fourths), secondary to cancer of some other organ, such as the stomach, uterus, the female breast, the rectum, or the vertebræ.[1] In more than one-third of the cases it is secondary to cancer of the stomach.[2] The symptoms of cancer in these various organs will

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[1] The nodular variety is most commonly secondary.

[2] According to Sir W. Jenner (Brit. Med. Journ. 1869, I., 205) cancer passes from

therefore materially aid the diagnosis. Even when the cancer is deposited first in the liver, other parts, such as the cœliac, mediastinal, inguinal, and cervical glands, and the lungs, are apt to become affected, and thus throw fresh light on the primary disease (see Case LXXXVII.). The diagnosis is also in many cases greatly assisted by the presence of a small mass of cancerous induration in the abdominal wall around the navel.

11. Cancer of the liver always runs a rapid course. The medullary cancer often grows very rapidly,[1] and is fatal within a few weeks or months; and although scirrhus is said sometimes to last for two years,[2] it is rarely prolonged beyond twelve months. The very fact of an enlargement of the liver having lasted much longer than this would be an argument against its being due to cancer.

12. The diagnosis is often assisted by the circumstances under which the disease occurs.

a. The age of the patient is sometimes of assistance in diagnosis. Cases are extremely rare where the liver is primarily affected with cancer before 35 or 40. Secondary cancer of the liver, it is true, may occur at any age, but then the primary disease will point to the nature of the case.

b. In a large proportion of cases there is no difficulty in tracing a history of cancer in the family. In the course of my practice I have known two sisters die of cancer of the liver, in one instance within a fortnight, and in another within a few months of one another.

c. In a large proportion of cases it will be found, whether a family taint can be traced or not, that the first symptoms of indisposition have been preceded by protracted grief or anxiety.

13. The diseases most likely to be mistaken for cancer of the liver are waxy disease, interstitial hepatitis or cirrhosis, syphilitic disease, catarrh of the bile-ducts, impacted gall-stone, multilocular hydatid, and pyæmic abscess (p. 224.) Case XCII. also shows how it might be possible to mistake cancer of the liver for even simple hydatid.

a. The smooth infiltrated form of cancer may be mistaken for waxy degeneration. In both there is a smooth, uniform, very hard enlargement of the liver; but in the waxy enlarge-

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the liver to the stomach oftener than in the opposite direction, but this is contrary to my experience.

[1] In one case Dr. Farre calculated that in ten days the liver acquired an addition equal to 5 lbs. Morbid Anatomy of the Liver, p. 28.

[2] Budd, Dis. of Liver, 3rd ed. p. 413.

ment the progress of the disease is slow, there is an absence of pain or of the cancerous cachexia, and there is usually also enlargement of the spleen, with albuminuria, and a history of constitutional syphilis, caries of bone, or protracted discharge from a suppurating surface; whereas in cancer there is no enlargement of the spleen or albuminuria, but the course of the disease is rapid, and there are pain, cachexia, and often signs of cancer elsewhere. Rare cases, where cirrhosis and waxy disease coexist (see pp. 32 and 47), may be mistaken for nodular cancer. In both there may be a nodulated hard enlargement of the liver with ascites. The points of distinction are the same as between the smooth form of waxy disease and cancer.

b. In cirrhosis the liver may be large, nodulated, and tender, and there may also be jaundice and ascites (see p. 139). It will be distinguished from cancer by the previous habits of the patient, a history of alcoholic dyspepsia with morning sickness, and the venous stigmata on the cheeks.

c. Syphilitic enlargement of the liver—either interstitial hepatitis with projecting gummata, or waxy liver indented by deep cicatrices—may be mistaken for cancer (see p. 147). In both affections there may be a large nodulated tender liver, with jaundice, ascites, and severe pain; but the syphilitic disease may often be distinguished by the comparatively early age of the patient, the previous history, and the existence of other evidences of syphilis.

d. Jaundice from catarrh of the bile-ducts, when it persists for several months, and is associated, as it may be, with nausea, retching and emaciation, may be mistaken for cancer (see p. 153). It is true that in catarrh of the bile-ducts there is little or no pain and rarely much enlargement of the liver. Still, when jaundice supervenes for the first time in an elderly person who is not the subject of gout or of constitutional syphilis (see p. 154), and is persistent, it is most probably due to cancer of the liver or in its vicinity, notwithstanding the absence of pain, vomiting, or any physical signs of tumour, and in any case this view would be favoured by a family history of malignant disease.

e. A gall-stone impacted in the common bile-duct may be mistaken for cancer of the liver. In both affections there may be intense jaundice with paroxysms of severe pain, vomiting, emaciation, and loss of strength. But in cancer the emaciation and failure of health precede for some time the pain and jaundice, whereas in gall-stone the patient has been in his usual health until his sudden seizure with biliary colic, and very often there is a history of previous attacks. In cancer the vomiting and pain may occur independently, whereas in cancer they are more commonly simultaneous. The concurrence of ascites could of course not be accounted for by gall-stones alone; but in a future lecture I shall have occasion to point out to you that gall-stones are not unfrequently followed by cancer of the gall-bladder and liver. (See also Case LXXXIX.)

Occasionally it will be tolerably clear from the symptoms that the patient is the subject of malignant disease, although the seat of the disease may be doubtful, whether it be in the liver or in some adjoining part, such as the stomach or omentum: but this is a difficulty of little moment as regards either prognosis or treatment.

f. A multilocular hydatid tumour of the liver may present all the clinical characters of cancer, viz. a hard nodulated tumour, intense and persistent jaundice, ascites, œdema of the legs, and rapidly increasing emaciation and prostration. Vomiting is a common symptom in cancer, but has rarely been observed in multilocular hydatid; whereas in the latter affection there is almost invariably considerable enlargement of the spleen, which Frerichs noted in only 12 out of 91 cases of cancer. A duration much in excess of twelve months would be opposed to cancer; but although multilocular hydatid has been known to last for years, in most cases its course is as rapid as that of cancer. It follows that an absolute diagnosis between the two affections would in many cases be impossible; but considering its rarity, multilocular hydatid is not often likely to embarrass the diagnosis (see Lecture VII.).

Treatment.—The treatment of cancer of the liver must be entirely palliative. There is no known remedy which can arrest or retard its progress. Mercury, iodine, arsenic, and the Sanguinaria Canadensis, which at different times have been recommended for the purpose, have been shown to be worse than useless. In none of the many diseases of the liver for which it has been the fashion to give mercury, has it been productive of so much injury as in cancer. The treatment must be restricted to supporting the patient's strength and nutrition by appropriate food, correcting errors in digestion, relieving pain, and procuring sleep.

1. The diet ought to be nutritious, but moderate and

digestible, and ought to contain a large proportion of the nitrogenous principles of food, and comparatively little of saccharine and oily substances which are calculated to increase the work thrown upon the liver. Alcoholic stimulants will often be necessary in the advanced stages of the disease, but ought to be given in moderation and well diluted. It must not be forgotten that an excess of nutriment or stimulants may feed the disease instead of nourishing the patient. In those hopeless cases where the primary disease is cancer of the stomach, the diet must consist mainly of milk and animal soups and jellies.

2. Various remedies will often be necessary to correct errors in digestion. For vomiting, bismuth, hydrocyanic acid, limewater, creasote, nux vomica, or ice, will be useful, and likewise the occasional application to the epigastrium of a sinapism or small blister; in the latter case, advantage is sometimes also derived from sprinkling over the blistered surface a quarter of a grain of morphia. The use of blisters for any other object can do little good, and may weaken the patient besides putting him to unnecessary pain. Flatulence will be relieved by the ethers and essential oils, but better still by such remedies as charcoal, creasote, or carbolic acid, which absorb the gas, or, by arresting decomposition, prevent its formation. A dose of from ten to thirty minims of a saturated aqueous solution of carbolic acid, with a few drops of chloric ether in peppermint water, is sometimes a most effectual remedy for this symptom. The bowels are often constipated, and will require relief, but care must be taken to avoid castor-oil and powerful purgatives, which will either nauseate the stomach or lower the patient by producing copious watery discharges. Four or five grains of the compound rhubarb pill with a grain of blue pill and a grain of extract of henbane, will usually produce the desired result satisfactorily and safely, or the bowels may be cleared out from time to time by a simple enema. The compound liquorice powder of the Prussian Pharmacopœia is also useful for the same purpose.

3. Sooner or later, in most cases, anodynes will be necessary to relieve pain or procure sleep. The hydrate of chloral, belladonna, conium, or Indian hemp, will often be found useful for these objects, and ought to receive a trial in the first instance; but in most cases it will be necessary to have recourse ultimately to one of the various preparations of opium or morphia. The solution of the bimeconate of morphia, which is of the same strength as laudanum, has less tendency to derange

the stomach or constipate the bowels than most other forms in which opium is given; and these disadvantages of opium will also be, in a great measure, avoided by the subcutaneous injection of morphia. In many cases I have known great relief obtained from a silvered pill containing one drop of creasote, quarter of a grain of extract of nux vomica, and from a sixth to half a grain of morphia, twice or three times daily. Lastly, poultices and warm fomentations, with or without a few leeches, may be required for intercurrent attacks of peri-hepatitis.

The following cases, which, with three exceptions, have been under your observation, illustrate the remarks that have now been made on cancer of the liver.

Case LXXXV.—*Cancer of Liver and Ovary—Jaundice, but no Ascites.*

Hannah C——, aged 50, a cook of large build and rather stout, married, a mother of one child, adm. into Middlesex Hosp. on July 28, 1863. She stated that for many years she had been subject to 'bilious attacks' (vomiting and headache), but that about two years before admission, these had become much less frequent and severe, and she had enjoyed good health until about ten weeks before admission, when she had been attacked somewhat suddenly with pain in epigastrium and right hypochondrium and in both shoulders, accompanied by great languor, and followed next day by diarrhœa, which lasted a week. A month before admission, pain had become much increased, and urine was noticed to be of a dark greenish-brown colour; a week later skin became yellow, and since then patient had suffered much from itchiness. From first she had been losing flesh.

The symptoms, while patient was under observation, were as follows. Skin, conjunctivæ, and serum of a blister of a bright orange colour, and great itchiness of entire surface. Tongue, at first clean, became afterwards coated with a thin white fur. At first there was no vomiting, but frequent attacks of nausea and a feeling of distension and oppression after meals. Appetite very bad. Motions destitute of any trace of bile, pultaceous, clay-coloured, and very offensive. Much pain in both shoulders and in epigastrium and right hypochondrium; this was much greater a few days after admission than subsequently. Liver much enlarged, hepatic dulness in right mammary line extending from ½ an inch below nipple to 1½ in. below ribs, and measuring 6¼ in.; portion of liver below ribs hard, tender, and distinctly nodulated (fig. 18, p. 209). No ascites. Urine scanty, only about one-half of normal quantity, sp. gr. 1030, acid, dark like porter, and threw down a copious deposit of lithates; it contained abundance of bile-pigment, but no bile-acids (by Harley's test), and no albumen.

Pulse 60; cardiac and respiratory signs normal, except that occasionally 'crackling sounds' were heard over base of right lung. On Aug. 6, and again on Aug. 15, it was noted that patient vomited after her medicine. On Aug. 29 there was a considerable increase of pain and tenderness in abdomen, with vomiting and pinched features. Under treatment, these symptoms abated somewhat, but vomiting returned from time to time, while the languor and prostration rapidly increased. On Sept. 28 vomiting became incessant, and from this date patient gradually sank until death on Oct. 3.

Treatment consisted in bismuth, hydrocyanic acid and opiates, sinapisms to epigastrium, and nutritious but digestible food.

Autopsy.—Body well nourished, and a thick layer of fat everywhere beneath skin, in omentum, and around kidneys. Tissues throughout body deeply stained with bile. No fluid in peritoneum, and no sign of recent peritonitis. Mucous membrane of stomach and intestines normal, but contents of bowel contained no trace of bile, and none could be squeezed from gall-bladder into duodenum. Liver very large, weighing 97 oz., and its right lobe measuring 13 in. from before backwards; surface studded with elevated yellowish-white, moderately firm nodules, varying in size from a pea to a walnut, and many of them depressed in centre. Similar masses seen in interior of liver on section; one mass, size of a large orange, occupied entire thickness of right lobe in front, extending back to transverse fissure, and in contact with upper surface of gall-bladder. These masses yielded a creamy juice on section, which contained characteristic 'cancer-cells;' some of them softened in centre into a yellow pulp, and here cancer-cells contained much oil, and there were many compound granular cells. Hepatic lobules between cancerous masses had a peculiar appearance; the central third of each lobule had a dark olive-green colour, and hepatic cells in it contained much bile-pigment; the outer two thirds were pale-yellow, and there the secreting cells were loaded with oil. Several stellate crystals of tyrosin were found in secreting tissue. Gall-bladder contained no bile, but was filled with facetted gall-stones. Hepatic ducts considerably dilated, but common duct passed into a mass of dense areolar tissue and enlarged glands in portal fissure, through which its continuity could not be traced. Capsule of liver at many places adherent by firm fibrous bands.

Uterus normal. Left ovary as large as a walnut, rather soft, and nodulated; it contained a little semi-fluid dark blood, and its substance was soft and yellow, and exuded a creamy juice containing 'cancer-cells.' A cancerous nodule, size of a pea, projected from surface of left ovary. Mesenteric and lumbar glands presented no abnormal appearance.

Lungs and heart normal, with exception of pulmonary congestion and patches of atheroma in mitral flaps and in commencement of aorta. No cancerous deposits in either spleen or kidneys.

CASE LXXXVI.—*Cancer of Uterus and Liver—Ascites, but no Jaundice.*

On Oct. 18, 1866, Charlotte D——, aged 56, was transferred to my care in Middlesex Hosp., having been for two months before under care of Obstetric Physician for cancer of uterus. She was married, and mother of nine children; catamenia had ceased at age of 49. Three years before she came under my care, she had an attack of what appeared to be gall-stones, sudden spasmodic pain in right side, with vomiting and slight jaundice, and ever since she had suffered from a feeling of uneasiness and fulness below right ribs. Twelve months before, she first noticed a slight but very offensive and persistent yellow discharge from vagina, and ever since she had suffered from costiveness and pain in defæcation and some difficulty in micturition. On two occasions, nine months and three months before she came under my observation, she had rather copious uterine hæmorrhage, lasting for about a fortnight. Two months before, she first noticed her abdomen to swell, and she began to suffer from vomiting after food. She had been losing flesh for twelve, and rapidly for three months.

On admission patient was weak and emaciated, and her countenance was expressive of pain. Extensive induration and ulceration of cervix uteri and upper part of vagina, with a fetid discharge. Abdomen much distended, measuring $35\frac{3}{4}$ in. at umbilicus, and exhibiting all the signs of fluid in peritoneum. Liver much enlarged, in r. m. l. measuring $6\frac{1}{2}$ in., and projecting fully 2 in. below costal margin; portion that could be felt hard and tender, but had no feeling of nodulation. Superficial abdominal veins slightly enlarged, but no jaundice. Tongue moist and slightly furred; vomiting had ceased, but bowels had not acted for several days. Urine loaded with lithates, but contained no albumen. No anasarca of trunk or extremities. Pulse 108 and feeble; no dyspnœa; cardiac and respiratory signs normal, with exception of slight dulness and fine crepitation at end of inspiration at base of right lung.

Patient was treated with bismuth and chloric ether, subcutaneous injections of morphia, mild laxatives, and a nutritious diet, with a small allowance of brandy. Vomiting did not return; but every night she suffered from intense pain in abdomen, which was only partially relieved by morphia injections. Belly slowly increased in size; prostration became daily greater, until death occurred on Oct. 30.

Autopsy.—Peritoneum contained several quarts of turbid serum, with flakes of soft lymph, chiefly on fundus uteri and in pouches before and behind. Cervix uteri entirely destroyed by cancerous ulceration, which extended for $1\frac{1}{2}$ in. down anterior wall of the vagina; lower two-thirds of uterus infiltrated with cancerous matter. Lumbar glands slightly enlarged from cancerous deposit, and in the portal fissure was a mass of enlarged cancerous glands pressing on portal vein. Liver of enormous size, weighing 115 oz., and portion opposed to thoracic and abdominal wall measuring 7 in.; it was studded throughout with numerous isolated nodules of cancer, from a pea up to a walnut in size, but none of them much raised above outer surface, so that portion of organ projecting beyond ribs was perfectly smooth and even. On section, many of nodules were found to be softening in centre into a flaky serous fluid. On microscopic examination, nodules, both at circumference and in centre, were seen to consist mainly of nuclear elements, with but few cells; hepatic tissue intervening between nodules free from cancerous infiltration. Mucous membrane of stomach and intestines healthy, but small nodules of cancer, up to size of a cherry, were scattered through lower lobe of right lung.

Although no opportunity was afforded for a post-mortem examination in the following case, the diagnosis, as I frequently pointed out in the wards, was sufficiently clear.

Case LXXXVII.—*Cancer of Liver, Lungs, and Cervical Glands—Jaundice and Ascites.*

John B——, aged 47, a cowman, adm. into Middlesex Hosp. Aug. 24, 1866. Twelve years before admission he had been confined to bed for a week with rheumatism; and two years before he had suffered for two months from severe pain at epigastrium, usually worse after food. With these exceptions, he had enjoyed good health until eight weeks before he came to hospital, when he was seized somewhat suddenly, while at work, with violent pain in region of liver and stomach, which never ceased, although it had been sometimes more severe than at others. Eight days after this he noticed that his motions had lost their colour, and that urine was very dark, and after six more days, conjunctivæ, and then skin, became yellow.

On admission patient was weak and emaciated, and had intense jaundice of entire surface. He complained of severe pain in region of liver, coming on in paroxysms, which would last for many hours, were sometimes attended by vomiting, and often prevented sleep. Liver enlarged, measuring 5½ in. in right mammary line; in epigastrium it felt hard and obscurely nodulated, and was very tender. No tumour felt corresponding to gall-bladder. There was neither ascites nor enlargement of abdominal veins or spleen. Tongue coated with a creamy fur; bowels costive; motions clay-coloured and very offensive; urine of colour of porter, and contained abundance of bile-pigment but no albumen. Pulse 96; cardiac and respiratory signs normal; no dropsy.

Patient was treated with mineral acids and gentian, anodyne draughts with drachm doses of tincture of henbane, and mild laxatives.

On Aug. 28 he first noticed a tumour on left side of neck, immediately above clavicle, about size of a hen's egg, hard, nodulated, and slightly tender. This tumour increased in size, and soon became seat of severe pain, like that in liver. Patient also complained often of severe pain down back, but no tenderness of spine. Indian hemp and henbane failed to give relief to these pains, and on Sept. 9 subcutaneous injections of morphia were resorted to, at first with great benefit. On Sept 5 ascites was first noticed, which from this date continued to increase, and on Sept. 24 both feet and lower half of both legs were noted as swollen and œdematous. Tumour in neck now filled up whole of lower triangle, and at its circumference were several large and movable glands quite distinct from general mass: patient vomited occasionally after breakfast, and became daily thinner and weaker. On Oct. 1 he was noted as vomiting almost everything he swallowed. Pulse 84, weak, and intermittent. Ascites and tumour of neck continued to increase; liver appeared larger and more distinctly nodulated; and pains were only relieved by morphia injections, which were repeated twice daily. No cough, and respiration slow and easy, but over middle of left lung posteriorly marked dulness over a space 3 or 4 in. square, with absence of vesicular murmur, but no friction or crepitation. On Oct. 5 left arm and hand œdematous, and vomited matter, which from first had resembled yeast, was found to contain abundance of sarcinæ. A mixture was ordered every six hours, containing ten minims of chloric ether and one drachm of a saturated aqueous solution of carbolic acid in peppermint water.

Patient was now so weak that he obviously could not live many days, but his wife came, and insisted on removing him to country.

CASE LXXXVIII.—*Primary Infiltrated Cancer of Liver—Great Enlargement, but surface smooth—No Jaundice or Ascites.*

On Jan. 5, 1876, Ann G——, aged 42, was sent to Samaritan Hosp., supposed to be suffering from ovarian disease. On Jan. 7 she was transferred to St. Thomas's. No history of malignant disease in family. Mother of eight children and had three miscarriages. Catamenia had ceased five years before, after birth of last child. Left eyeball had been removed 3½ years before, owing to effects of a blow. Six months before, when apparently in perfect health, first noticed swelling below right ribs which gradually filled abdomen, and at same time she lost flesh and strength. For two months had suffered much pain in abdomen, and for five days had noticed swelling of legs and thighs.

On admission: Very emaciated. Abdomen greatly enlarged, bulging abruptly forwards below ribs; enlargement greater in upper than in lower part; integuments stretched and shining. Girth at navel 33½ in., and half-way between this and sternum 33 in.; from ensiform cartilage to navel 8¼ in.; from navel to pubes 6½ in. No sign of fluid in peritoneum, and swelling evidently caused by a very large liver, lower margin of which can be felt on both sides, 2½ in. below level of umbilicus; margin of right lobe more rounded than that of left, and the two separated by a deep indentation rising to above navel. Upper margin of liver does not rise too high in chest; total hepatic dulness in r. m. l. 11 in., and same in mesial line. Surface of swelling slightly undulating, but free from excrescences, and not tender Consistence tense, but rather elastic. Suffers much from constant tightness in tumour, worse after meals and preventing sleep, and has occasionally attacks of severe 'scraping' or cutting pain. Tongue coated; no appetite; no vomiting; bowels costive; no jaundice. Urine 1026, loaded with lithates; no albumen. Pulse 96. A few dry bronchial râles over lungs.

Became rapidly weaker; vomiting came on; and died on Jan. 13.

Autopsy.—Liver greatly enlarged, corresponding to tumour observed during life; weighed 198 oz.; shape normal; non-adherent; surface smooth; left lobe as large as an ordinary right; great exaggeration of central fissure. Enlargement of liver due to extensive cancerous infiltration of left lobe and to a considerable extent of right; on section a few isolated nodules, from size of a pin's head to an inch in diameter; but none projected from surface. No cancer in any other part of body. Spleen 7 oz., dark and soft; lungs congested. On section of infiltrated new growth in liver it was pale like a fatty liver, outlines of acini being distinct. Microscopic examination showed that it had ordinary structure of encephaloid cancer.

The two following cases are remarkable for the mode of commencement. In Case LXXXIX. the disease seemed to commence in the gall-bladder and bile-ducts, and the history in the first instance pointed to biliary colic and gall-stones rather than to cancer; while in Case XC., where the disease perhaps originated in the right kidney, one of the first symptoms of illness was ascites.

CASE LXXXIX.—*Cancer of Gall-bladder, Bile-ducts, Liver, &c., commencing with severe pain like that of Biliary Colic—Jaundice—No Ascites.*

Anne G——, 63, adm. into St. Thomas's Hosp. Nov. 19, 1875. No history of malignant disease in family. Had seven children, all of whom, as well as husband, had died of consumption. Had been of temperate habits. Had suffered now and then for a year or two from flatulence, but with this exception she had been in her usual good health until three weeks before admission, when one day, while out washing, she had been suddenly seized with a sharp shooting pain below right ribs in front. Pain was so severe that she could scarcely walk home. It kept coming on in paroxysms, and was attended by shivering but not by vomiting; after four days it ceased, but a day or two after this she was noticed to be deeply jaundiced, and she had much nausea. On cross-examination patient admitted that for two or three months before attack of pain strength had failed a little, but she had followed her work as usual, and had not noticed any loss of flesh.

On admission, deeply jaundiced; skin very itchy; urine loaded with bile-pigment, but none in stools; hepatic dulness not increased; slight bulging and some tenderness corresponding to gall-bladder; no ascites; no appreciable abdominal tumour. Tongue clean; fair appetite; pulse 70.

Four days after admission had an attack of severe pain in liver, lasting about half an hour. These attacks recurred first at intervals of a few days and then more frequently; they were not attended by vomiting. Day by day patient grew weaker and thinner, and there was loss of appetite and much nausea. The liver gradually increased in size, until, on Jan. 22, it measured 7 in. in r. m. l. No inequalities could be felt on its surface; no tumour appreciable anywhere, but always much tenderness over gall-bladder; no ascites. Death by exhaustion on Jan. 26.

Autopsy.—No fluid in peritoneum. Colon and duodenum drawn up and adherent to an irregular cancerous mass projecting from liver, occupying situation of gall-bladder, infiltrating adjacent part of liver and extending downwards so as to involve head of pancreas. Immediately above pancreas another cancerous mass due to infiltration of glands in that region. Duodenum where adherent to mass narrowed, but mucous membrane both of it and colon healthy. Gall-bladder shrunken; walls $\frac{1}{4}$ in. thick, infiltrated with cancer; inner surface rough, shaggy, and very vascular; cystic duct obliterated; a small rounded orifice surrounded by granulations at fundus where it had been adherent to abdominal wall. Walls of common and of hepatic duct from $\frac{1}{4}$ to $\frac{1}{2}$ in. thick from infiltration with new growth, this infiltration extending both downwards towards bowels, and upwards for 4 in. into substance of liver, where it became obliterated and embedded in hard new growth. No gall-stones. On cutting into liver, bile-ducts greatly dilated behind seat of stricture, forming cystiform sacculations filled with glairy, colourless fluid, free from all colour of bile. Scattered through substance of liver were numerous nodules of new growth; and in centre of many of them a small orifice from which a drop of glairy fluid exuded on squeezing, as if they were formed by infiltration of walls of bile-ducts. Portal vein not obstructed. Weight of liver 75 oz. Spleen $6\frac{1}{2}$ oz. Commencing granular degeneration of kidneys; in upper lobe of right lung a circumscribed new growth, size of a walnut; lower lobe studded with new growths from size of a pin's head to that of a pea.

CASE XC.—*Cancer of right Kidney, Liver, Spleen, and Lungs. Ascites the first symptom of illness.*

John M——, aged 37, adm. into St. Thomas's Hosp. March 9, 1875. No evidence of malignant disease in family. Habits temperate; no history of syphilis, and general health good. For three or four months before Christmas 1874 had felt occasionally a fulness and tightness of abdomen, but had not paid much attention to it. On Dec. 28 had a more severe attack than usual of this tightness, and after this it became more constant and was increased after food. Still appetite continued good; no nausea or vomiting; no appreciable emaciation; and followed employment as a platelayer until Feb. 28, when he was suddenly seized with great tightness and pain in abdomen; could take no food; and was compelled to give up work and take to bed. From this date abdomen continued to enlarge, and he was slightly yellow.

On admission, sallow and anæmic, and conjunctivæ slightly yellow. Still complains much of tightness in abdomen and of dyspnœa on exertion. Girth at umbilicus 36 in.; moderate ascites; slight œdema of legs. Liver much enlarged, dulness commencing ¼ in. below right nipple, and from this to lower margin, 3 in. below ribs in r. m. l. 7 in.; enlargement uniform; surface smooth, hard, and painless; left lobe also much enlarged. Splenic dulness increased; and lower end of spleen felt projecting 1½ in. below ribs. Abdominal veins slightly enlarged. Tongue clean: appetite fair; no vomiting; bowels confined. Complains much of painful tightness of abdomen after food. Pulse 78; heart signs normal. Lungs normal. Urine 1017; no albumen, but some bile-pigment. Temp. 99°.

The treatment at first consisted in the perchloride of mercury and bark, with aperients; while mercurial and belladonna ointment was applied to abdomen, and morphia was given occasionally to relieve pain and procure sleep. On March 12 two small excrescences were discovered on surface of liver, one just below ensiform cartilage and the other on left lobe. On March 31 a mixture of nux vomica and acid was substituted for the mercurial. On April 12 patient had gained 9 lbs. in weight in ten days and 12 lbs. since admission, but this increase was probably due to greater accumulation of fluid in abdomen, which now measured 40 in.; parietes attenuated and glazed; more œdema of legs; jaundice scarcely appreciable. April 19: Seven pints of fluid drawn off by aspirator, reducing girth to 35 in. and giving great relief. Fluid rapidly accumulated again; much pain in abdomen; occasional epistaxis. On April 28 girth of abdomen again 40 in.; patient's weight 16 lbs. more than on admission. On May 7, 16 pints of serum removed by paracentesis, and after this liver could be seen forming a large prominent tumour between sternum and umbilicus—hard and nodulated. Pain again relieved by operation, but exhaustion increased, and death on May 13.

Autopsy.—Eight pints of serum in peritoneum. Liver greatly enlarged; weighed 161 oz.; studded with cancerous nodules, many of which projected from surface. Portal vein much dilated, and on tracing it into liver cancerous masses were found moulded to shape of veins and branching with them, but not adherent to their walls. Gall-duct pervious; spleen, 17½ oz., contained several masses of cancer. Right kidney entirely destroyed, being simply a bag of soft disintegrating cancer. Vessels of right kidney compressed by cancerous nodules projecting from liver, and veins extremely dilated. Left kidney much enlarged, but healthy; stomach and pancreas healthy. Lungs congested and studded with cancerous masses.

The next case to which I shall direct your attention was remarkable not only for the early age of the patient and the rapid course of the malady, but more particularly for the pyrexia which marked its course. Little is known as to the range of temperature in cancer. Wunderlich makes the following observations upon the subject. 'It is a peculiarity of cancer cases that elevated temperatures are comparatively rare, and that the temperature generally maintains itself on a normal, or even subnormal, plane, which, however, by no means precludes the occurrence of high temperatures through intercurrent complications, or at the close of the disease. But fever temperatures of long duration are at least rare in cancer patients.' In confirmation of this opinion, Dr. Woodman, the translator of Wunderlich's treatise, quotes cases observed by Drs. Finlayson, Da Costa, and E. B. Baxter, and adds: 'The few observations I have myself made of carcinoma of the liver, uterus, and breast, before marasmus had set in, only show very slight elevations of temperature, or none at all; never above 101° Fahr. unless from some complication; whilst I have found subnormal temperatures with rapid pulse in several cases of advanced cancer with emaciation.'[1]

My own experience coincides with the opinions now quoted, and, I believe, with those of most observers, viz.:—that in cancer, unless there be some inflammatory complication, the bodily temperature is at or about the normal standard,[2] and accordingly in the case of any obscure internal disease, a continuous elevation of temperature would in itself be opposed to the diagnosis of cancer. But the case now related proves that this rule is not absolute. In this case, moreover, the age of the patient, 24, was opposed to cancer of the liver; while not only the pyrexia, but the rigor, the previous injury and enlargement of the testicle, the rapid course, and the cerebral symptoms, all favoured the diagnosis of pyæmic inflammation, in preference to cancer of the liver.

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[1] On the Temperature in Diseases, by C. A. Wunderlich, Syd. Soc. Transl. 1871, pp. 429, 430.

[2] Since this case occurred, I have met with another case, a lady aged 56, with primary uncomplicated cancer of the liver, and a temperature of 102°.

Case XCI.—*Acute Cancer of Liver with Pyrexia in a man aged* 24.

James C——, 24, carpenter, adm. into St. Thomas's Hospital Nov. 6, 1872. No history of malignant disease in family, and previous health good. Six months before, strained himself whilst turning a crane; left testicle swelled and was tender, but general health appeared unaffected. Six weeks before admission first complained of pain in right side of abdomen and began to lose flesh and strength. Soon after a doctor discovered a swelling in right hypochondrium, which rapidly increased. Had no rigors, but twice during sleep had perspired profusely.

On admission, emaciated; hectic flush on cheeks; temp. 101·3° F. Suffered much from pain in region of liver, and from dyspnœa. On right side of abdomen was a visible bulging, continuous apparently with liver, its lower margin extending almost to brim of pelvis, and upper margin of hepatic dulness reaching to 1½ in. below nipple; surface of swelling firm, smooth, and moderately tender. Distinct jaundice of skin and conjunctivæ; no ascites nor enlargement of abdominal veins. Tongue moist, slightly furred; no appetite; much thirst; no vomiting; bowels open but not loose; bile in stools. Urine 1018; contained copious lithates, some bile-pigment, and a trace of albumen. Lay on right side; respirations 32, thoracic; some cough, but no expectoration; sibilant râles over both lungs, and over back of both lungs, but chiefly on right side, breath sound feeble and slight impairment of resonance on percussion. Pulse 120; heart's sound normal. Left testicle twice size of right, hard but not tender.

Patient was ordered a milk diet and an effervescing saline mixture. He had also morphia draughts and subcutaneous injections of morphia, and laudanum poultices to relieve pain, but he got rapidly worse. Nov. 8.—Pulse 134. Vomited last night a greenish flocculent matter containing no food. Nov. 11.—Has repeatedly vomited green bilious matter, and jaundice is now very decided. Liver increased in size, more bulging below ribs, and its dulness extending to within ½ in. of nipple; surface smooth and firm. Tongue red and dry; bowels open daily. Very prostrate, and occasionally delirious. No rigors or night-sweats. Pulse 120; a systolic bruit audible at base of heart and propagated upwards to clavicles and neck. Nov. 13.—Delirium, jaundice, and enlargement of liver increased. Swelling below ribs is more elastic, but there is no distinct fluctuation. Yesterday had a decided rigor, followed by heat and perspiration. Nov. 15.—Much more prostrate, but no more shivering. Skin dry. Still delirious. Got rapidly weaker, and died on Nov. 16.

The following is a note of observations of the temperature:—

his life he had been very intemperate, and he had been in the habit of drinking a large quantity of spirits. For some months he had been losing flesh, and he had been suffering from occasional nausea and other dyspeptic symptoms, and from pains in epigastrium. Dr. Greenhow discovered that liver was enlarged and distinctly nodulated below margin of right ribs, and recognised the peculiar physiognomy characteristic of the cancerous cachexia. There was no jaundice, and little or no ascites; and nothing was observed to indicate an immediate fatal termination.

On Aug. 26 the patient was brought to hospital, and admitted under Dr. Goodfellow, his condition having become suddenly worse about two days before. His symptoms on admission were great prostration and cachectic countenance; marked jaundice of skin, conjunctivæ, and urine; complete loss of appetite, urgent vomiting, intense pain and tenderness in region of liver, which was much enlarged, hard and nodulated; abdomen much distended and fluctuating: small, rapid pulse.

No improvement took place; and day after admission patient vomited a large quantity of dark bloody-looking fluid.

During night of 27th he fell into a state of collapse, which continued until death at 2.30 P.M. of August 28.

Autopsy.—Moderate emaciation; marked jaundiced tint of conjunctivæ and skin and of tissues generally, including the internal organs and bones. Between five and six quarts of dark-red bloody serum in peritoneal cavity, and lying on upper surface of right lobe of liver, towards its right extremity, between it and diaphragm, was a dark coagulum of blood which weighed 5 oz. avoirdupois. Serous coat of intestines, which was bathed by the bloody fluid, presented no abnormal injection or deposit of lymph. Liver weighed 72 oz.; right lobe was relatively much enlarged, measuring 9 in. transversely, while left lobe was much atrophied, and a mere appendage to right, not exceeding 1½ in. in its transverse diameter; greater part of diminutive left lobe granular on surface, and presented on section appearances characteristic of cirrhosis. Corresponding to lobus quadratus was a rounded mass, about size of a large walnut, attached by a narrow pedicle, and likewise composed of cirrhotic glandular tissue. Whole surface of right lobe of liver covered with prominent nodules, varying in size from a pea to a large cherry, the largest being very elastic or almost fluctuating; they were most developed near anterior margin of right lobe on upper surface. The coagulum on surface of right lobe was adherent at one spot near right extremity of organ, corresponding to one of softened nodules, which was ruptured. The structure of right lobe of liver was extremely dense; and on making a section, it appeared to consist of two abnormal elements, a groundwork of firm grey scirrhus-looking tissue, infiltrated with a creamy yellowish juice, and containing a number of cavities up to size of a cherry, filled with a soft pulpy bright yellow substance; whole of right lobe appeared to be made up of these abnormal elements, and scarcely presented at any part a trace of natural glandular tissue or of bile-ducts. The scirrhous structure had encroached to some extent along anterior margin of left lobe.

On examining with microscope juice scraped from denser scirrhous portions, it was found to contain a multitude of rounded, elliptical, and fusiform cells, up to $\frac{1}{800}$ of an inch in diameter, with one or sometimes two large nuclei about one-third size of cell; many of cells, likewise, included brownish pigment-granules. In softened portions, similar cells were discovered, mixed up with a large quantity of oily and pigmentary matter, both inside and outside cells.

Other abdominal organs normal. Heart normal. Apices of both lungs condensed and puckered, and contained encysted calcareous masses up to size of a pea.

In Case XCIV. the immediate cause of death was also probably hæmorrhage into the peritoneum. The preparation which I show you appears to be an illustration of that rare form of disease described by Dr. Bright and other writers as 'fungus hæmatodes' of the liver, where the growth projects greatly from the general surface of the organ. The transition between the secreting cells of the liver and the large cells of the growth, determined by myself and Dr. Cayley, is likewise a matter of considerable pathological interest.

CASE XCIV.—*Cancerous Tumour (Fungus Hæmatodes), projecting from upper surface of Liver—Hæmorrhage into Peritoneum.*

Luke T——, aged 57, was sent to London Fever Hospital on Jan. 20, 1868, supposed to be suffering from 'fever.' He had no friends, and could give no account of his previous history. On admission he had a heavy stupid countenance, and his mind was confused. He was very emaciated; pulse varied from 76 to 88, and was very weak. Tongue dry and brown; bowels rather loose; abdomen slightly distended, partly from tympanites, but partly also from fluid in peritoneum. Hepatic dulness appeared to be normal. Occasional cough, with thin frothy expectoration; slight dulness over both lungs posteriorly, with rather fine crepitation, but no tubular breathing. No night-sweats; neither jaundice, dropsy, nor albumen in urine.

Patient was treated with ammonia, and subsequently with iron and mineral acids, along with beef-tea, milk, and brandy; but symptoms became gradually worse, emaciation and ascites increased; frequent low muttering delirium; and on Feb. 2 slight jaundice, but motions still contained bile. Pulse rarely exceeded 80. Patient became every day weaker, but no fresh symptom of importance appeared. He died on *Feb. 16.*

Autopsy.—Peritoneum contained between three and four quarts of dark sanguinolent fluid. Liver was separated from diaphragm in front and from anterior abdominal wall for 2 or 3 in. by a space filled with this sanguineous fluid ; suspensory ligament elongated in a corresponding degree. Liver weighed 64 oz. ; capsule slightly thickened and opaque, but surface smooth. On section it appeared unusually dense and tenacious. Projecting from upper and back part of right lobe was a rounded tumour as big as a man's fist. This was embedded in a hollow in diaphragm, to which it was so firmly adherent that part of it was left behind in removing liver. This tumour was of pulpy softness, and reflected over it was the thickened capsule of liver, from inner surface of which the pulpy mass could be easily scraped with handle of knife. On section there was seen to be a sharp line of separation between it and the dense tissue of the rest of liver. The pulpy substance was torn with greatest facility, and was very vascular, so that it was obviously the source of blood in peritoneum. On microscopic examination, it was found to be made up of large nucleated cells, with an average diameter of $\frac{1}{300}$ inch, or about three times that of a hepatic gland-cell. The cells were rounded, pyriform, or caudate, and each contained one or sometimes two nuclei, with much fine granular matter ; some were full of oil-globules, and some contained brown pigment-granules exactly like what is seen in gland-cells of liver. Along with these large cells, which were much the more numerous, were others of smaller size, and not to be distinguished from secreting cells found in other parts of liver (fig. 19).

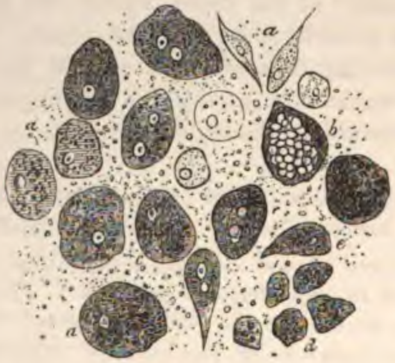

Fig. 19. Shows microscopic appearances of tumour of liver in Case XCIV. *a*, Large nucleated cells of various shapes, and some with a double nucleus ; *b*, similar cells containing oil-globules ; *c*, large cell containing bile-pigment ; *d*, cells resembling in every respect glandular epithelium of liver ; *e*, transitional forms between these last cells and the large cells.

Stomach and intestines healthy ; walls of heart thin and soft ; both lungs firmly adherent, and much congested in dependent parts. Nothing noteworthy in any other organ.

Case XCV. is an illustration of cancer implicating the liver, without producing any symptoms or signs which could lead to its existence being suspected during life. One of the suprarenal capsules also was destroyed by cancer, and yet there was no vomiting or bronzing of the skin. It is now well known that the suprarenal capsules may be destroyed by cancer, without any of the symptoms of Addison's disease resulting, so that these symptoms must be ascribed, not so much to the destruction of the capsules, as to the morbid process by which this is effected.

CASE XCV.—*Cancer of Vertebræ, Suprarenal Capsule, Liver, and Lung—No symptoms of Disease of Liver.*

Alfred T——, aged 55, admitted into Middlesex Hosp. under my care, Jan. 28, 1868. He was very weak and emaciated, and not very connected in his replies. Seventeen years before admission he contracted syphilis, followed by constitutional symptoms, but his 'present attack' commenced only three months before admission with severe pain in spine, accompanied by emaciation and weakness.

His symptoms while under observation were as follows:—Progressive emaciation and debility, and anæmic chlorotic colour of face; but no jaundice, or bronzing of skin, or discoloration of mucous membrane of mouth, or perspirations. Persistent pain and tenderness on pressure over spinous process of third and fourth lumbar vertebræ, but no sign of tumour or of paraplegia, excepting retention of urine for last two or three weeks of life. Tongue dry, red, and fissured; no vomiting; constipation alternating with diarrhœa. Abdomen distended and tympanitic, with slight tenderness on deep pressure to left of umbilicus: a few days before death abdominal swelling subsided, and aorta could be felt passing along spine, but no appreciable tumour. Hepatic dulness 4 in. in r. m. l. At no time was there tenderness, or a feeling of nodulation in region of liver, or ascites. Pulse varied from 84 to 120, always small and weak; cardiac dulness diminished; at no time any cough or expectoration, and at time of admission no notable sign of mischief could be discovered in lungs. Urine alkaline, and contained phosphates, but no albumen or bile-pigment. Temperature either normal, or but slightly increased. Throughout mind was confused, and there was a tendency to low muttering delirium, increasing towards death, which occurred on March 22.

Autopsy.—A soft cancerous tumour of bodies of third and fourth lumbar vertebræ, projecting about half an inch from surface, chiefly on left side, where it invaded texture of psoas muscle, and encroaching about half-way to the spinal canal, which, as well as the spinous processes, appeared normal. Cancerous enlargement of lumbar and bronchial glands, and a mass of soft cancer, size of a large walnut, compressing a large branch of pulmonary artery in upper part of lower lobe of right lung. Liver not enlarged, and its lower margin did not project beyond edge of ribs, but it contained from a dozen to twenty isolated cancerous nodules, from size of a pea to that of a walnut, several of which were excavated in centre. One of these nodules was in a portion of liver which was firmly adherent to right suprarenal capsule; latter organ greatly enlarged, and converted into a mass of hard cancer, measuring 2½ in. in diameter. Left capsule, kidneys, and brain presented nothing abnormal.

The following case came under my notice while I was House Surgeon in the Edinburgh Royal Infirmary. It is an example of a rare form of cancer implicating the liver, but causing no symptoms of hepatic disease.

CASE XCVI.—*Melanotic Cancer of Penis, Lymphatic Glands, Liver, Pleura, etc.*

James L——, aged 54, a butler, was admitted into Royal Infirmary, Edinburgh, on Feb. 4, 1851. He was a tall, robust man; hair was dark brown, and eyeballs were remarkable for their prominence, and for a bluish tint of sclerotics. Attached to lower and outer surface of prepuce, and extending a considerable way along its free margin, was a tumour, size of a chestnut, of a dark brown, almost black colour, its surface nodulated and covered with a fetid, dirty yellow, puriform discharge. When pricked with a pin, it bled profusely, and it was often the seat of acute pain, especially during, and for a short time after, micturition. It had been growing for two years, and had commenced as a small black wart on the outer surface of prepuce, about an inch from its free margin; this wart for six months remained stationary, but afterwards increased more rapidly. On reflecting prepuce, which was done with some difficulty, there were displayed on surface of glans several warty excrescences of a bluish-black colour, and varying in size from a pin's head to half a pea. In each groin was a swelling of size of a hen's egg, which had first appeared about three months before admission.

For three months patient had complained of dyspnœa and cough; and on examining chest, left side presented a uniform bulging, measuring fully 1 in. more in circumference than right; there was also on this side marked dulness on percussion, imperfect expansion, and absence of respiratory murmur and of vocal thrill. Apex of heart displaced to left margin of sternum. Physical signs of right lung normal. Pulse 90, very feeble.

After this, patient got rapidly worse; he lost all relish for food, and became very prostrate. The fits of dyspnœa increased in frequency and in severity, lasting sometimes for several hours, and dulness with suppression of respiratory murmur was observed over base of right lung. Tumour on penis and swellings in groins increased slightly in size. No jaundice, ascites, or enlargement or pain of liver.

On morning of March 26 he had an unusually severe attack of dyspnœa; pulse 84, and almost imperceptible; extremities cold; face livid and eyeballs more prominent. These symptoms continued until death on evening of 27th.

Autopsy.—Tumour on penis presented on section a smooth black surface, yielding on section a copious inky juice. Lumbar, inguinal, and femoral glands enlarged and infiltrated with black matter; and some of them entirely converted into a pulpy black fluid. Lymphatics of spermatic cord contained one or two small melanotic nodules. Along whole of abdominal aorta was a chain of enlarged glands. Some of these exhibited, on section, a black pulpy mass; while others, which were but slightly enlarged, presented normal glandular structure, with circumscribed brownish-black points. Hypogastric and sacral lymphatics normal.

Left pleura distended with several quarts of fluid tinged with blood and black pigment, which pushed apex of heart towards right side. Scattered over whole of parietal and pulmonary pleura were masses of a dark deposit, varying in size from smallest appreciable point to half an inch in diameter, and, for most part, presenting a circular outline; largest of these nodules projected about one-sixth of an inch from surface of pleura; smallest were not appreciably elevated, presenting a punctiform appearance not unlike shading of a chalk drawing. The large nodules were almost black, while punctiform deposit had a brownish-black tint, tinged more or less with purple. Most of nodules were covered by epithelial layer of pleura, but at back part of cavity, where they were confluent and aggregated into flattened masses, this membranous lining was at some places wanting, and masses exhibited a pulpy irregular surface, and yielded on pressure a large quantity of dark juice very like liquid sepia. Left lung compressed and carnified; at reflection of pleura from root of lung upon ribs was a layer of recently extravasated blood, at some parts half an inch in thickness. Right pleura contained a few ounces of fluid similar to that in left; and its surface exhibited nodules of deposit of same character, but less extensive. Embedded in substance of right lung were a few circumscribed black nodules, the largest about size of a cherry: around them, pulmonary tissue was normal and crepitant. Bronchial glands were all black, but not much enlarged; in posterior mediastinum glands were greatly enlarged, and a cluster of them, forming a mass, size of an orange, was situated in angle of bifurcation of trachea, in front of œsophagus; deep cervical glands contained black pigment.

Between mucous and muscular coats of œsophagus were one or two rounded nodules, size of a barley-corn, containing black pigment; rest of alimentary canal and mesenteric glands normal. On surface of liver were seen about a dozen nodules of black deposit, about one-third of an inch in diameter; numerous similar masses embedded in substance of organ, which was but slightly increased in size. In spleen was a single mass of black deposit, size of a pea. Kidneys contained in cortical substance several melanotic nodules, size of a swan-shot. Between muscular and mucous coats of bladder and of urethra were a few black nodules, size of barleycorns.

Chemical Examination of Melanotic Matter.—The following analysis of the pigmentary matter was made by the late Dr. James Drummond:—

'It was insoluble in water, alcohol, and ether. When treated with hydrochloric, nitric, and sulphuric acids, it was dissolved; the solution being nearly colourless. When chlorine gas was passed through it suspended in water, it was bleached to a certain extent, but not entirely. When boiled with potash, it dissolved, with disengagement of ammonia. The ultimate analysis yielded the following result:—

Carbon	67·01
Hydrogen	6·45
Nitrogen	11·45
Oxygen	8·36
Ash	6·73
	100·00

'The ash consisted, in great part, of peroxide of iron.'

Microscopic Examination of Melanotic Matter.—The dark juice from tumour on penis contained a large quantity of granular matter of a sienna-brown colour; granules were solid and angular, and refracted light strongly; acetic acid produced no change upon them, but strong nitric acid rendered them much lighter. Mixed up with these granules were a few nucleated cells, having a circular or oval outline, and a diameter of about $\frac{1}{500}$ of an inch. Some of cells were more elongated, and one or two exhibited a caudate appearance. Most of them were loaded with coloured granules, which quite obscured all appearance of a nucleus. In some of cells, however, which contained little or none of coloured granules, one and sometimes two nuclei could be detected, with one or two distinct nucleoli. When a small particle of tumour was torn out with needles and examined, it exhibited a network of fine filamentous tissue, infiltrated through meshes of which were the elements of the dark-coloured juice just described. The melanotic deposits in pleura and in lumbar and inguinal glands were subjected to careful microscopic examination, and were all found to possess a structure similar to that of tumour on penis.

LECTURE VII.

ENLARGEMENTS OF THE LIVER.

SPINDLE-CELL SARCOMA—MYXOMA—EPITHELIOMA—CYSTO-SARCOMA—MULTILOCULAR HYDATID—SIMPLE CYSTS—TUBERCLE—LYMPHATIC GROWTHS—ENLARGEMENT WITH XANTHELASMA—ENLARGEMENTS OF GALL-BLADDER.

GENTLEMEN,—In this lecture I purpose to bring under your notice certain diseases which occasionally lead to enlargement of the liver, but which are of comparatively rare occurrence, and as to the clinical history and diagnostic characters of which our knowledge is as yet imperfect.

XII. SPINDLE-CELL SARCOMA OF THE LIVER.

The following case is an example of a form of enlargement of the liver hitherto (1873 [1]) undescribed. It illustrates the importance, on clinical grounds, of distinguishing the anatomical characters of the different lesions still too commonly grouped under the common designation of 'cancer.' Until within the last few years, the disease in this case would, from a structural point of view, have been regarded as a variety of cancer. Structurally, it is now acknowledged by pathologists to be distinct from cancer, while it will be seen that the clinical history of the patient was very different from that of true cancer of the liver. 1. There was no evidence of the so-called cancerous cachexia. The patient had never the appearance of a man suffering from malignant disease; four months before his death his weight was exactly the same as it had been twelve months before, although all this time the disease in the liver had been progressing; and he continued to go about and follow his employment until within two or three weeks of his death, the cause of which was obscure. 2. Considering the size of the tumour, there was much less pain than might have been expected on the supposition that the disease was true cancer.

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[1] *The case* was communicated to the Pathological Society, Jan. 21, 1873.

The severe, but rare and transient, attacks of pain in the right side were more like what might have been expected to result from the calculus found after death in the right kidney, than from the disease in the liver. For a time a burning pain was complained of in the liver, but for many months before death this had quite ceased, and latterly the chief complaint was a feeling of tightness due to the size of the tumour. 3. There was neither jaundice nor ascites. 4. The similarity in structure between the tumour of the eyeball and that of the liver pointed to a constitutional origin; but the interval between the primary and secondary lesion was much greater than in true cancer, unless we are to suppose, what would have been equally incompatible with cancer, that the disease had been going on in the liver for eight or nine years, without giving rise to symptoms, until it produced an appreciable tumour. 5. Primary cancer of the liver is not common at so early an age as that of my patient. It remains to be seen whether these clinical characters will hold good in other cases of tumour of the liver presenting the same anatomical structure.

It is to be regretted that the post-mortem examination was far from being complete, and in particular that it failed to account for the patient's somewhat sudden death.

CASE XCVII.—*Spindle-cell Sarcoma of Liver.*

Mr. L. N——, aged 30, first consulted me at my house on Oct. 9, 1871. He stated that 18 months before he had been attacked with a sharp pain between right ribs and ilium. The pain came on in severe paroxysms, but after two days it ceased; it was not attended by vomiting, nor followed by jaundice. Nine months afterwards he had a second similar attack of about the same duration. For two months he had been losing flesh, but not to a great extent, and one month before he came to me Dr. Brown of Whitchurch had found the liver to be considerably enlarged, and in the interval this enlargement had much increased. He was a man of very temperate habits, and had never had syphilis. On examination, I found a tumour filling the right side of abdomen to within two inches of pubes, continuous upwards with liver, percussion dulness of which ascended as high as nipple in front, but not too high behind. Behind tumour, in both flanks, there was tympanitic percussion sound. The tumour formed a perceptible prominence in right side of abdomen, and right lower ribs bulged out considerably. At umbilicus girth of right side of abdomen was 16¼ in., and of left, 15¾ in., and girth of chest, 2 in. below nipple, was 17 in. on right side, and 16½ in. on left. The surface of the tumour was uneven from the presence of several semi-globular elevations; its consistence was doughy, especially over most prominent parts, but there was nowhere any feeling of fluctuation, vibration, or elasticity, or any tenderness on pressure. The patient complained of a frequent burning pain in tumour, which often kept him awake at night, and of a feeling of weight after meals; but he did not suffer from nausea or vomiting; his appetite was good, and bowels regular, and he had not lost strength. He could walk five or six miles a day without fatigue. Urine deposited a copious sediment of lithates, and became almost black on addition of nitric acid after boiling; but it contained no albumen, nor did it exhibit ordinary reaction of bile-pigment with nitric acid. Heart was pushed up, its apex being felt between fourth and fifth ribs, just below nipple.

An opinion had already been expressed by several physicians who had been consulted that the tumour was hydatid, but this view was negatived by:—1. The absence of any fluctuation or elasticity in the prominences on its surface; 2. Its rapid growth; 3. The burning pain; and, 4. The patient's statement that on April 2, 1862, his left eyeball had been excised by Mr. Hulke for what had been called 'a malignant tumour.' On the other hand, it seemed clear from its consistence that if the tumour was cancer it must be a rapidly-growing *soft* cancer, and this view was negatived by:—1. The healthy appearance and strength of the patient; 2. His good appetite and but slightly impaired digestion; 3. His family history. His father and mother were both alive and well, and no member of his family had suffered from cancer; 4. His age; 5. The long interval of good health between excision of eyeball and commencement of disease in liver. The opinion given to the patient was that the tumour was something more solid than hydatid, and that no benefit would be derived from paracentesis. As the tumour appeared to be of an unusual nature, I wrote to Mr. Hulke to ascertain the nature of growth in eyeball removed in 1862. Not regarding my opinion as satisfactory, the patient went on the same day to Sir W. Gull, whose opinion was that the tumour was not hydatid, and probably cancer. By the same post which brought my letter, Mr. Hulke received another from Sir W. Gull, making a similar enquiry.

Mr. Hulke had fortunately preserved copious notes and microscopic drawings of tumour in eyeball. For two years before patient had consulted him in March 1862 there had been a progressive decrease of the visual field in left eye, and for three months complete loss of sight. At first there had been no external signs, but for one month there had been redness and œdema of the conjunctivæ and intense pain. On consulting Mr. Hulke the man looked healthy, except that left eyeball was distended and hard, and pupil widely dilated and motionless; iris was discoloured and pushed forwards, and at temporal side of fundus oculi could be seen a solid buff-coloured tumour, advancing nearly to lens, and covered by retina and choroid. After enucleation, a tumour was found in the choroid in the situation observed during life. It was greyish, and on section there exuded a viscid yellowish, rather than a creamy juice. It consisted mainly of small fusiform nucleated fibre-cells (fig. 20), the prolongations of which were woven into a tangled web, whose meshes were filled with a hyaline albuminoid matrix. Mr. Hulke added that in accordance with the views then held, the tumour was called a medullary cancer, but that 'its structure was characteristic of what we now, in Virchow's terms, call a spindle-cell sarcoma.' The man made a rapid recovery, and there was never any return of tumour in the cicatrix.

On obtaining this information, I wrote to the patient's medical attendant, Dr. Brown of Whitchurch, expressing the opinion that the tumour of liver, like that of eyeball, was probably a spindle-cell sarcoma, and that the case was one of unusual interest.

The patient continued to follow his employment as an upholsterer, and I heard nothing more of him until June 10, 1872, when he again came to London to consult me. The tumour had increased in size, girth at umbilicus being 34 instead of 32 in., and upper margin of hepatic dulness in front having risen to above nipple. It extended across middle line as far as left lumbar region. At many places, especially those which were most prominent, it felt much more tense and elastic than it had done previously, but nowhere was there any distinct fluctuation or vibration. In beginning of April patient had experienced a third attack of severe spasmodic pain below right ribs, but this had ceased after the use of chloral and subcutaneous injections of morphia. He was now also free from the burning pain of which he had complained eight months before. As long as he was quiet he had no pain whatever, but when he moved much, or stooped in his business, he had a good deal of pain below right ribs. He also suffered from dyspnœa on exertion, and a feeling of fulness after meals. At the same time patient did not look any worse than when I had first seen him, and his weight was exactly the same as it had been twelve months before. His tongue was clean and appetite good; no jaundice, no ascites, and no enlargement of abdominal veins. He was still following his business, and he could walk an hour without fatigue.

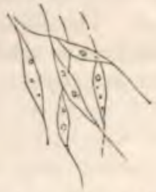

Fig. 20. Group of spindle-cells from tumour of choroid, × 240. From a drawing by Mr. Hulke.

Although the circumstances of the case now pointed somewhat more to hydatid, the same opinion was expressed to patient as before;

but as he was very desirous to have something done, he was told that no harm could result from an exploratory puncture, which would remove all doubt on the matter. I advised, however, that he should previously have the advantage of a consultation with Sir W. Jenner, who accordingly saw the patient with me on June 12, and who concurred in the difficulties of the case, and in the advisability of solving them by paracentesis. A small trocar was accordingly introduced into the most elastic portion of the tumour below right ribs; only a few drops of blood came away, which exhibited nothing but blood-corpuscles under microscope. No bad effect followed puncture, and in a few days patient returned to his home and resumed his business, which he continued to follow until Oct. 8, the tumour slowly increasing. On Oct. 8 he had a severe attack of spasmodic pain over whole surface of tumour, which was relieved by application of hot-water bags and repeated doses of hydrate of chloral. He continued, however, to suffer from a feeling of tightness, due to presence of the tumour, and, experiencing no relief, he left his home on Oct. 16 to try the effect of hydropathy. He had throughout his illness consulted a great many medical men, both in London and in the provinces, and even taken the opinion of a female clairvoyante respecting his case. On Oct. 25 he died at the Turkish Baths, Bristol. All that I could learn of the symptoms which preceded death was that for 36 hours before he had suffered from intense pain over the tumour to right of the umbilicus, which was somewhat relieved by subcutaneous injections of morphia. The pain was unattended by vomiting.

The body was examined by Dr. T. D. Nicholson, of the Turkish Bath establishment, Bristol, to whom I am indebted for the following particulars, and for sending to me portions of the diseased structures for examination.

The peritoneum was adherent at several places to surface of enlarged liver, but there was no recent lymph. Projecting from anterior surface of each lobe of liver was a rounded soft, apparently cystic tumour, about 2 in. in diameter, and pressing against the abdominal wall. Projecting from under surface of liver, and intimately connected with it, was an enormous mass of morbid structure, composed of cyst-

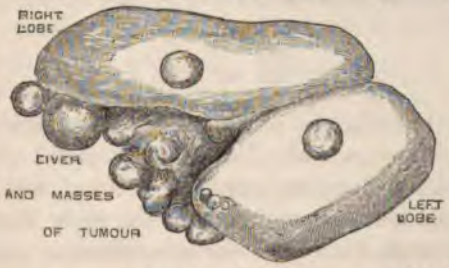

Fig. 21. Shows tumours projecting from liver. From a sketch by Dr. Nicholson.

like bodies, varying in size from that of a cherry to that of a child's head (fig. 21). This mass, together with the liver, weighed 20 lbs. and 1 oz. avoird. The contents of the small tumours on upper surface of liver were dark grey and gelatinous; those of the large masses on under surface were of lighter colour and of pultaceous consistence. Some of lumbar glands were as large as beans, and contained a soft grey material. Spleen and kidneys were healthy, except that right kidney contained a dark rough calculus about size of half a walnut. Heart and lungs were normal.

The portions of the tumour forwarded to me were submitted to Mr. Henry Arnott for microscopic examination. After hardening in a solution of chromic acid, the gelatinous material of which the tumours were composed was found to be a typical example of spindle-cell sarcoma, as will be seen by the annexed drawing by Mr. Arnott (fig. 22).

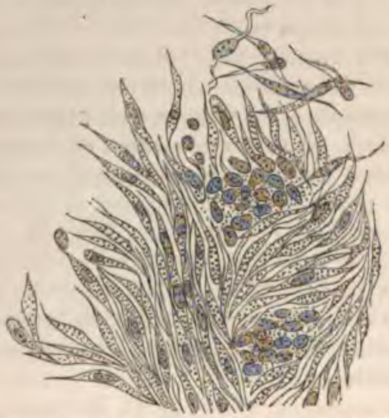

Fig. 22. Represents a section of the growth from the liver, × 220. From a drawing by Mr. Henry Arnott.

XIII. MYXOMA OF THE LIVER.

The clinical characters of this growth in the liver are not yet sufficiently known. Mr. Nunn has recorded a case in which a tumour of this nature, as large as a fœtal head of the full period, occupied the posterior part of the right lobe of the liver and projected from its upper surface, where it was closely adherent to the under surface of the diaphragm. The patient was a female aged 38, who within twelve months of her death had been twice operated upon for a recurrent myxoma of the breast.[1]

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[1] Path. Trans. 1872, vol. xxiv. p. 120.

XIV. EPITHELIOMA OF THE LIVER.

In the 25th volume of the 'Pathological Transactions,' Dr. Greenfield has described a case of primary columnar epithelioma of the liver. The patient was a female aged 33, whose clinical history was identical with that of cancer. It is not improbable that in many cases of 'cancer' of the liver the new growth has a similar anatomical structure.

XV. CYSTOSARCOMA OF THE LIVER.

Naunyn has recorded a case in which the liver was found studded with small tumours having a structure similar to that of Cystosarcoma of the mamma. The liver, which was of normal size and shape, was obtained from the body of a female aged 62, who had been for a long time under the care of Professor Frerichs and whose chief symptom was marasmus.[1]

XVI. MULTILOCULAR OR ALVEOLAR HYDATID TUMOUR.

This is a very rare form of tumour; only about eighteen cases have been recorded, not one of which has occurred in this country. It is composed of numerous minute hydatids contained in cavities interspersed through a hard, almost cartilaginous, matrix of fibrous tissue, and not, as in the case of an ordinary hydatid, enveloped in a parent cyst. Its clinical characters are very different from those of an ordinary hydatid tumour of the liver. It forms a rounded tumour, varying in size from that of a hen's egg to twice that of a man's head, and in most cases is situated in the right lobe. When small and deeply seated it may be inappreciable during life. In most cases it can be felt, or it causes a distinct bulging. The tumour is not smooth, elastic, fluctuating, and painless, but nodulated, hard, of even cartilaginous consistence, and tender. In almost all cases there is considerable enlargement of the spleen, and early supervening intense jaundice. Ascites is somewhat less common than jaundice, and in many cases there is œdema of the legs in the advanced stage. Progressive emaciation and prostration and deranged digestion (but not vomiting) are constant symptoms, while attacks of partial peritonitis and hæmorrhages are not uncommon towards the end. In many cases the tumour ultimately suppurates in the centre and induces symptoms of hectic fever. The disease for which multilocular hydatid would be most readily mistaken is cancer, and like this it sometimes runs a rapid course of a few months (see p. 214); but in other cases it has been known to exist for ten years and more prior to death. The treatment recommended for ordinary hydatid tumours is obviously inapplicable here, and our efforts must be limited to the relief of symptoms as they arise.[1]

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[1] Reichert und du Bois Reymond: Archiv 1866.

XVII. SIMPLE CYSTS OF LIVER.

Several writers have described simple cysts in the liver, containing a clear watery fluid or thick mucus. They are usually very numerous and of small size, the largest not exceeding that of a hazel nut; and they are sometimes associated with similar cysts in the kidneys.[2] I know no case, however, in which cysts of this sort have enlarged and suppurated, so as to be distinguishable during life. In the following case the precise nature of the disease was obscure. During life the diagnosis was suppurating hydatid. It was clear that there was suppuration somewhere. A circumscribed empyema was negatived by the history, and by the fact that the fluctuation was below and not above the solid liver. Opposed to tropical abscess was the fact that the patient had never been out of England, nor suffered from dysentery; while pyæmic inflammation never leads to the large collection of pus which was obviously present. For suppuration outside the liver no cause such as disease of the spine or ulcer of the stomach could be discovered. In fact all causes other than hydatid appeared to be excluded; and the appearance of the cysts at the post-mortem examination was believed at first to confirm the diagnosis made during life. But the failure to find any trace of hydatid structure in the contents of the cysts rendered this view scarcely tenable, for even in a sterile hydatid some trace of the parent membrane would have been present. Failing hydatid, we are reduced to view the case as one either of primary abscess or of suppurating cysts; and in this difficulty it is much to be regretted that the intestines were not carefully examined. As to abscess, there are not only the objections already referred to, but the appearance of the wall of one of the cysts was such as could scarcely have resulted from a recent inflammation, and it seems almost inconceivable that three abscesses should form in the liver in immediate contact with each other, the wall of one forming part of the wall of the other, and that one of these should project as a cyst from the surface of the liver, without any sign of inflammation of the superimposed peritoneum. On the other hand, although large cysts are not uncommon in the kidneys, they have not, so far as I know, been hitherto described in the liver. On any view of the case, the symptoms resulting from the bursting of one of the cysts into the peritoneum, and particularly the absence of pyrexia, are interesting.

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[1] The most complete account of multilocular hydatid tumours will be found in the two following memoirs,—De la Tumeur Hydatique, Alvéolaire, par le Dr. J. Carrière, Paris, 1868: and Étude Chir. sur la Tumeur Échinocoque multiloculaire du Foie, par le Dr. Ducellier, Paris, 1868.

[2] Frerichs, Dis. of Liver, Syd. Soc. Ed. ii. 223.

CASE XCVIII.—*Suppurating Cysts in Liver—Rupture of one cyst into Peritoneum. Acute Peritonitis—Pyæmic Abscesses in Liver and Lungs.*

Harriet C——, aged 43, adm. into St. Thomas's Hosp. June 18, 1875. Father 75, mother 76, both alive and healthy; a sister of mother died of phthisis; three brothers and five sisters, all alive, 34 to 55, and well, except one brother who has delicate lungs. Married; five children; two died in infancy. When quite a child, patient had a severe attack of inflammation of lungs, and ever since had been delicate and apt to catch cold. At age of 30 had a scaly eruption over body, which disappeared after eight weeks. Had never been out of England. Present illness commenced about 20th April with loss of appetite and strength, emaciation, sleeplessness, thirst, and occasional sickness. After a fortnight first felt pain in region of liver and right shoulder, not constant or severe except when she lay on right side or moved about. Four weeks before admission she became much worse; intense thirst, skin hot and dry in daytime, profuse sweating at night, and rapid emaciation.

On admission, very prostrate and emaciated; hectic flush on cheeks; profuse perspiration at night, or whenever she sleeps. Temp. 102° to 104°. Pulse 108. Heart's sounds healthy. Tongue too red, and dryish; no appetite; thirst; bowels open. Still much pain in right side, increased greatly by slightest movement. In right side of abdomen is a prominent swelling, reaching down to an inch below umbilicus and forming a distinct prominence between that and right ribs, which do not bulge; dull on percussion, the dulness continuous with that of liver, which reaches to $\frac{1}{4}$ in. below right nipple; total dulness in r. m. l. 10 in. Surface of swelling smooth, tender, soft and elastic, with a thrill as if from fluid. No evidence of fluid in peritoneum; no enlargement of abdominal veins; no jaundice; no œdema of legs. Posteriorly, hepatic dulness extends about two inches above normal level. Urine contains neither albumen nor bile. Sleeps badly.

Patient was ordered quinine, mineral acids and four oz. of brandy. On June 19 an exploratory puncture was made below right ribs with fine trocar; a few small beads of thick yellow pus oozed out. A few hours later a large trocar was introduced at same spot; only about a drachm of thick yellow pus came out. The silver cannula was fastened in and the opening covered with oakum. The opening gave great relief, but on 21st she had a rigor lasting twelve minutes, followed by a feeling of sinking and by friction and moist sounds over front of right lung. Elastic tubing was substituted for silver cannula. Thick pus continued to ooze slowly from tube, which at no time contained any trace of echinococci. On 23rd, upper margin of hepatic dulness had receded to fully an inch below nipple. A second slight rigor. On 24th, tympanitic distension of abdomen, but no tenderness. Frequent retching. Temp. 99. Pulse 128. Increased prostration. Notwithstanding opiates, these symptoms persisted with occasional hiccough. On June 28 had a sensation of profound sinking and thought she was dying, but rallied. On June 30 it was noted that upper margin of hepatic dulness had receded to 1½ in. below nipple, while lower margin of tumour had ascended, so that total dulness in r. m. l. was only 7½ in. On July 3 this was reduced to 6½ in.; no sign of fluid could be discovered in tumour, but there was clear evidence of fluid in peritoneum. Abdomen not tender and temperature normal. After this became daily weaker, was occasionally delirious, and aphthæ appeared on tongue, and feet more œdematous. On July 9 had a slight convulsive seizure, and on 12th she died. After June 24 temperature was never elevated and was often subnormal, once as low as 96°.

Autopsy.—Intestines greatly distended with gas. Transverse colon adherent along its anterior border to abdominal wall by somewhat firm adhesions, so as to form a sort of septum dividing peritoneal cavity into an upper and lower portion. Lower portion contained about 7 pints of turbid serous fluid, mixed with a large quantity of thick yellow curdy pus, which coated and was adherent to coils of small intestine. The fluid had not appearance of ordinary peritoneal exudation, but looked like a mixture of thick pus with it. Anterior surface of liver connected by soft adhesions to abdominal wall; corresponding to external opening adhesions were firm and fibrous, and sinus passed through them directly backwards into liver. Lower border of liver rather firmly adherent to colon, which slightly overlapped it, its edge not reaching much below normal level. Between adhesions which united hepatic flexure of colon with abdominal wall and under surface of liver was an irregular cavity of some size, full of thick curdy pus, and communicating with lower portion of peritoneal cavity by an oblique sinus passing down through layers of adhesion. Several circumscribed abscesses, about size of a marble, were seen on surface of liver, but no cyst was visible before removal of organ. On removal, greater part of right lobe was found to be elastic and fluctuating, and upper surface was convex, pushing diaphragm considerably upwards. On under surface of right lobe, near anterior margin, was a collapsed cyst, about size of an orange, with a smooth thick fibrous wall, which communicated by two small apertures in its under surface with abscess-cavity above referred to formed by adhesions on under surface of liver. It was this abscess-cavity which had apparently been opened during life. Further back in liver and closely adjacent to collapsed cyst was another, of about same size and projecting from surface; and above this was another larger cyst, forming projection on upper surface of right lobe, but covered by a thin layer of hepatic tissue. Both these cysts contained a thick curdy puriform fluid, their inner surface was irregular and ragged, and larger one opened into a sort of cavernous structure, formed apparently by multiple abscesses in substance of liver. In none of cysts could any trace of hydatid membrane, echinococci, or hooklets be discovered by naked eye or on most careful microscopic examination; gall-bladder contained normal fluid bile. Spleen 6 oz., firm and dark. Kidneys rather small, but structure normal. Intestines not carefully examined. Heart small but healthy. Right pleura contained 14 oz. of slightly turbid serum; surface of right lung rough from recent lymph, and several small pyæmic infarcti in middle and lower lobes. Two or three similar infarcti in lower lobe of left lung.

XVIII. TUBERCLE OF THE LIVER.

Enlargement of the liver is sometimes due to the deposit of tubercle. The subject of tubercle of the liver has still to be investigated. Rokitansky speaks of hepatic tubercle occurring 'in the shape of semi-transparent, greyish, crude, miliary granulations, in which case it is more especially the product of acute tuberculosis.'[1] Frerichs also mentions the occurrence of nodules of yellow tubercle in the liver, which may soften into vomicæ; while other observers have noted contractions and dilatations of the fine bile-ducts from the deposit of tubercle in their walls.[2] Enlargement of the liver occurring in the course of general tuberculosis may be due to tubercle, as well as to fatty or waxy deposit. There are no symptoms by which the tubercular enlargement can be distinguished during life, and its discovery would not materially modify either the prognosis or the treatment. In the following case the liver was infiltrated with minute miliary tubercles; the jaundice was apparently due to concurrent catarrh of the biliary passages, which was subsiding before the patient's death.

[1] Path. Anat., Syd. Soc. Transl. vol. ii. p. 149.

[2] Frerichs, Dis. of Liver, Syd. Soc. Ed. ii. 22

CASE XCIX.—*General Tuberculosis—Enlargement of Liver from Tubercular Deposit—Jaundice from Catarrh of Bile-ducts—Embolism of Spleen.*

Mary C——, aged 40, adm. into Middlesex Hosp. under my care, Dec. 17, 1867. Father and mother had both died at age of 50, of some chest affection, and of eleven brothers and sisters all were dead but one, though patient could not say of what they had died. Patient was extremely prostrate, and somewhat confused in her mind. So far as her history could be obtained, it was to the effect that six months before she had lost her appetite, and had vomited about half an hour after every meal. Two or three months after this she became jaundiced. She had not suffered from cough, hæmoptysis, rigors, or night-sweats, but from first she had lost flesh and strength.

On admission, jaundice of moderate intensity of skin and conjunctivæ; urine exhibited reaction of bile-pigment; copious deposit of lithates; no albumen. No itchiness of skin; tongue dry and brown, except at edges, which were preternaturally red. Patient stated that up to time of admission she had vomited almost everything within half an hour of swallowing it, but she did not vomit once after admission. A motion passed soon after admission was formed, and of a dark brown bilious colour. Hepatic dulness increased, in right mammary line measuring 5 inches, and extending fully an inch below margin of ribs; portion below ribs smooth and slightly tender. Pulse 120, small and feeble; a faint systolic bellows murmur at left apex of heart; temperature 100°·2. Nothing to attract attention to lungs, which in patient's weak state were not examined. A large superficial bed-sore over sacrum.

Patient was treated with bismuth, chloric ether, and stimulants, but she became rapidly more prostrate; low muttering delirium set in, motions and urine were passed in bed, and death took place on Dec. 23.

Autopsy.—A pint of clear serum in peritoneum. Liver very large; weighed 77 oz.; capsule not thickened or adherent; surface generally smooth, but marked by numerous minute depressions and elevations; glandular tissue pale yellow and opaque, exactly like that of a fatty liver, from which it differed, however, in being remarkably firm and tough. On section, a little thin watery bile could be squeezed from the divided bile-ducts, many of which presented small dilatations. Gall-bladder contained a small quantity of a similar fluid, as well as numerous minute, black, gritty concretions. On microscopic examination, it was ascertained by Dr. Cayley that enlargement of liver was due to presence of numerous miliary tubercles scattered through glandular tissue between lobules, and presenting all structural characters of grey tubercle, some of which were just visible to naked eye as minute grey specks. Mucous membrane of stomach pale, but immediately below pylorus that of duodenum, for about 8 inches, intensely injected, tumid, and studded with numerous small granular punctated elevations, apparently enlarged solitary glands. Lining membrane of common bile-duct also very red, and mucous membrane slightly swollen, but passage not obstructed. Three small tubercular ulcers in lower part of ileum. Both lungs studded with numerous grey miliary tubercles, and near both apices, a small patch of old grey tubercle. Edge of one of flaps of mitral valve much thickened. No lymph at base of brain, and no tubercles in pia-mater, but much serous fluid beneath arachnoid and in cerebral ventricles; in cavity of arachnoid over both hemispheres was a thin film of extravasated blood. In uterus a fibrous tumour as large as a cocoa-nut, and position of right ovary was occupied by a tumour as large as an orange, partly solid, and partly breaking down into a soft cheesy material. Right Fallopian tube as large as a finger, and filled with soft putty-like material; its lining membrane rough and ulcerated, like that of pelvis of kidney in tubercular pyelitis. Spleen large; weighed 15½ oz.; very soft, and studded with numerous abscesses, from a pea to a hazel-nut in size, and containing thick yellow pus; also several solid deposits in spleen, having the characters of recent infarcti. Cortices of both kidneys studded with minute yellow tubercular granules.

XIX. ENLARGEMENT OF LIVER FROM LYMPHATIC GROWTHS.

In leukæmia, and in cases where there is a general tendency to enlargement of the lymphatic system independent of leukæmia, the liver may be found studded with new formations, sometimes minute and greyish-white, not unlike miliary tubercles, at other times opaque yellow and as large as a cherry. Structurally these formations resemble lymphatic tissue, and they are believed to be developed in connection with the lymphatic system. When very numerous they may produce enlargement of the liver. In any case, therefore, of leukæmia, or of general enlargement of the lymphatic glands, enlargement of the liver may be due to this cause or to simple hypertrophy (see p. 54); but it will call for no special treatment apart from that of the general condition.[1]

XX. ENLARGEMENT OF THE LIVER WITH XANTHELASMA.

The following case was a well-marked example of the disease first described by Dr. Addison and Sir W. Gull in the seventh volume of the second series of the 'Guy's Hospital Reports,' under the name of 'vitiligoidea,' and which Mr. Erasmus Wilson subsequently designated 'xanthelasma.' In the nineteenth volume of the 'Pathological Transactions' two cases were recorded by Dr. C. Hilton Fagge, who in a summary of our then existing knowledge of the subject made the following statement. 'The nature of the change in the liver in this affection is as yet entirely unknown. So far as I can ascertain, a post-mortem examination has as yet been made in no case of vitiligoidea. The liver seems to be greatly and uniformly enlarged. No tubera or nodules have been felt on its surface' (p. 443).

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[1] For illustrations of this disease in the Liver, I must refer to Cases reported by me in the Pathological Transactions. Vol. xx. pp. 192, 198; and vol. xxi. p. 372.

Patches of vitiligoidea plana, such as existed in Angelo S——, are occasionally met with in the eyelids, independently of jaundice or of any obvious disease in the liver, or may, as Dr. Church[1] has pointed out, be sometimes hereditary, but this does not detract from the interest of the fact that in many instances the morbid change in the skin is associated with jaundice and enlargement of the liver, presenting peculiar clinical characters. The jaundice, as Dr. Fagge remarks, is usually of no ordinary kind. It is peculiar in being very chronic and persistent, lasting for months or even years, and although very decided in its hue, in being in most cases (but not invariably) independent of complete obstruction of the hepatic duct, bile being usually found in sufficient quantity in the alvine evacuations. The enlargement is distinguished by being great and uniform, and by its surface being firm, smooth, and somewhat tender.

In the following case the enlargement was found to be due to an excessive formation of fibroid tissue, and the case appeared to be an example of interstitial hepatitis, such as I have referred to in a former lecture (p. 139). Everywhere along the portal canals and between the lobules the liver was pervaded by a dense, firm deposit, made up of fibrous tissue and masses of minute corpuscles or nuclei. The glandular tissue of the liver was cut up by this dense deposit into circumscribed patches or islands, just as we see in a cirrhotic liver. The view that the case was one of enlarged cirrhotic liver is confirmed by the man's having been addicted to drinking spirits in great excess. But, on the other hand, the jaundice was greater and more persistent than is usual in cirrhosis, and there is no mention of

[1] Barth. Hosp. Rep. vol. x.

intemperance in most of the recorded cases of enlargement of the liver accompanying vitiligoidea.

The cause of the jaundice was not very apparent. The bilious motions seemed to show that it was independent of any obstruction of the large bile-ducts, while its duration and the absence of any symptoms of blood-poisoning until shortly before death were opposed to the view that it was due to a morbid state of the blood. The vitiligoidea itself, however, indicated a very disordered state of the patient's nutrition, so that the jaundice may possibly have been due to the bile-pigment, which is being constantly reabsorbed, not being sufficiently transformed into urinary pigment and other materials, by which in health it is eliminated from the body; or possibly it may have been occasioned by partial occlusion of the bile-duct from the pressure of enlarged glands in the fissure of the liver.

Case C. was the first in which the condition of the enlarged liver so commonly associated with xanthelasma was carefully examined after death.[1] Similar appearances were subsequently found in another case by Dr. Hilton Fagge.[2] But it has been now conclusively proved that very different affections of the liver may lead to the development of xanthelasma. In a subsequent lecture I shall relate to you a case in which it resulted from protracted obstruction of the bile-duct by a gall-stone. A similar case has been reported by Dr. Pye Smith;[3] while Drs. Wickham Legg and Duckworth have observed it in jaundice caused by obstruction of the hepatic duct by a hydatid,[4] and Dr. Moxon has seen it follow obstruction of the duct by simple stricture.[5] It would appear that this peculiar condition of the skin is apt to be developed in any case of jaundice, if sufficiently protracted.

CASE C.—*Enlargement of Liver from Interstitial Hepatitis—Jaundice—Xanthelasma.*

Angelo S——, aged 41, a paper-dealer, admitted into Middlesex Hospital, July 14th, 1868. Father and mother both dead; he could give no information respecting cause of their death. For many years he had been very intemperate, drinking large quantities of rum, brandy, and gin; and for four or five years he had suffered a good deal from indigestion and occasional vomiting, especially in morning. Two years before admission vomiting had become more constant, and always occurred directly after taking food. About same time patient had become weak and languid; he had lost appetite, and had noticed abdomen to swell. Six months later jaundice set in, and had never since disappeared. Twelve months before admission he had begun to suffer pain in region of liver and between shoulders, and six months before admission he had first noticed pale discoloured patches on eyelids. During last few months he had had occasional epistaxis, and now and then had passed a little red blood per anum, although he was not aware that he had piles. Motions had always presented a bilious yellow character.

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[1] The Case was reported in Path. Trans. 1869, vol. xx. p. 187.

[2] Ib. 1873, vol. xxiv. p. 242.

[3] Ib. vol. xxiv. p. 250.

[4] Ib. vol. xxv., p. 155 and St. Bartholomew's Hosp. Rep. vol. x. p. 60.

[5] Ib. vol. xxiv. p. 129.

On admission weak and emaciated, and obliged to keep bed. Whole surface of body and conjunctivæ deeply jaundiced. On both eyelids of both eyes were light cream-coloured patches, contrasting strongly with surrounding dark jaundiced tint. Lower eyelid of right eye almost completely involved in this discolouration, and in other lids patches varied from size of a pin's head to about half an inch in diameter. Patches had a smooth satiny feel; edges were well defined, and they appeared slightly raised above surrounding surface, but were really not so. No white patches on the gums, and no tubercular prominences on hands or elsewhere. Front of chest and neck were also variegated by large patches of brownish discolouration, running into one another, darker than surrounding jaundiced skin, and scaly on surface, like pityriasis. Circumscribed circular patches of a similar nature, about size of a sixpence, on both forearms near wrists. These brown patches were seat of considerable itchiness, from which skin generally was free. Abdomen greatly distended, owing mainly to enlargement of liver; girth at umbilicus 33½ in.; at lower margin of ribs it was 35½ in. Vertical hepatic dulness measured 6 in. in anterior mesial line, 9 in. in right mammary line, and 10 in. in axillary line, where it reached down to crest of ilium. Liver was seat of constant pain, and portion which projected beyond ribs was smooth, dense, and slightly tender; lower margin sharp and hard. Spleen much enlarged; its lower margin distinctly felt projecting three inches beyond margin of left ribs. No ascites, and no enlargement of superficial abdominal veins. Tongue moist, and coated with a white fur. Still occasional vomiting of food. Bowels regular; motions formed, and well coloured with bile-pigment. Urine contained much bile-pigment and a trace of albumen, but no sugar, leucin, or tyrosin; specific gravity 1012. Pulse 84; cardiac dulness slightly increased; a systolic bellows-murmur heard distinctly over third left costal cartilage. Slight dulness, with feeble breathing, over lower and back part of right lung.

After admission vomiting became worse, but was relieved for a time by pills containing creasote and morphia. It continued, however, to recur until death, and on August 23rd and 24th patient vomited a considerable quantity of sanguineous liquid. He had also repeated attacks of diarrhœa, motions always containing bile, and occasionally a little red blood. Tongue was usually dry and brown. Liver was always seat of much pain, and very tender, but no material change took place in its size while under observation, although as patient lost flesh enlargement of liver became more apparent; its margin could be distinctly seen through abdominal parietes moving up and down with respiration. Jaundice became darker and assumed somewhat of a bronzed hue, but conjunctivæ became less yellow, and there was no increase or alteration of light-coloured patches on the eyelids. Urine was examined on several occasions for leucin and tyrosin, but neither was found; usually, but not always, it contained a trace of albumen. Patient continued to lose flesh, and became greatly exhausted. About end of August he began to suffer from hiccough, restlessness, and delirium. When thwarted in the least he kept shouting at top of his voice, so that it was necessary to remove him to the delirious ward. On Sept. 4th aphthæ were noticed on tongue and on roof of mouth; diarrhœa recurred, and motions were passed in bed. On Sept. 19th teeth and tongue became coated with sordes, and on Sept. 20th the man died.

Autopsy.—Sections were made with a Valentin's knife through cream-coloured patches in eyelids *in situ*. The colour was found to be due to deposit in meshes of cutis of a large quantity of oily granules, both isolated and aggregated in masses, and always most abundant in neighbourhood of hair-follicles, which in other respects, however, appeared normal. When stained with carmine a distinct nucleus could be seen in centre of the oily masses. The epidermal cells were not affected. The oily matter was in such quantity in cutis that it poured out like a milky fluid from a section placed in water.

A few ounces of fluid in peritoneum. Liver very large, its lower margin reaching to below umbilicus; it weighed 80¾ oz.; outer surface smooth, capsule thickened, and at many places attached to surrounding parts by firm old adhesions; structure firm, and on section presented a smooth dense surface of a pale greyish colour, with very little appearance of ordinary secreting tissue, but apparently a great increase of fibrous element. Here and there were darker islands of a jaundiced tint, which seemed to be remains of secreting tissue. On microscopic examination denser material, of which greater portion of liver was composed, was found to consist of nuclear and fibroid tissue in portal canals and between lobules; nuclear element was particularly abundant; groups of rounded nuclei, or bodies resembling lymphatic corpuscles, were aggregated round minute vessels between lobules. In some parts of dense structure the nuclear, and in others the white fibrous tissue, predominated. At some places nuclei seemed club-shaped or oat-shaped, and appeared to pass into fibres; this appearance, however, was quite exceptional. Yellow portions of liver were found to be made up of hepatic lobules, cells of which were loaded with oil and bile-pigment. Green bile was found in duodenum, and bile could be squeezed with ease from gall-bladder along bile-duct into bowel. Glands in fissure of the liver along spine, and along course of iliac arteries, were much enlarged and jaundiced. Spleen greatly enlarged; weighed 23 oz.; capsule much thickened, and in its interior numerous opaque yellow embolic deposits up to size of a large pea. On posterior wall of stomach, a few inches from pylorus, was a simple ulcer, size of a crown-piece, base of which was formed by tissue of pancreas; its surface had a glazed cicatrized appearance, and there was slight puckering of edges. Both kidneys large; they weighed together 10¾ oz.; right contained numerous opaque yellow embolic deposits, one of them as large as a walnut; left kidney contained two or three similar but smaller deposits. Heart was not hypertrophied; it weighed 11¼ oz.; aortic valves competent, but two of them grown together, and their point of attachment to aorta partially separated, so that they seemed to form one valve; several small rough vegetations on their ventricular surface.

XXI. ENLARGEMENTS OF THE GALL-BLADDER.

The gall-bladder may be enlarged from various causes, so as to form a tumour attached to the liver, and appreciable through the abdominal parietes. The causes, the symptoms, and the treatment of these enlargements will be considered in a future lecture. (Lecture XIII.)

LECTURE VIII.

CONTRACTIONS OF THE LIVER.

SIMPLE ATROPHY—ACUTE OR YELLOW ATROPHY—CHRONIC ATROPHY—(CIRRHOSIS—SIMPLE AND SYPHILITIC INDURATION—RED ATROPHY).

GENTLEMEN,—In previous lectures I have described to you the normal limits of the area of hepatic dulness (p. 2), as well as the principal causes of apparent and real enlargement of the liver, with the means of recognising them. We have now to consider the chief causes of a diminution in the area of hepatic dulness, and their distinctive characters. And in the first place you must remember that the area of hepatic dulness often appears diminished, although the organ in reality retains its normal weight and bulk.

SPURIOUS CONTRACTIONS OF THE LIVER.

The main conditions likely to induce an apparent diminution in the size of the liver are as follows:

1. Tympanitic distension of the bowels, and particularly of the transverse colon and stomach, may prevent the lower margin of the liver being felt, and diminish the area of hepatic dulness in several ways:

a. A portion of stomach or intestine distended with gas may become interposed between the surface of the liver and the abdominal parietes.

b. When the lower margin of the liver is thin, and when there is excessive tympanitic distension of the subjacent bowels pushing the liver forwards and rendering the abdominal parietes tense, the lower edge of the liver may escape detection on palpation, and its dulness on percussion may be imperceptible.

c. In excessive tympanites the antero-posterior diameter of the abdominal cavity is increased, and the lower portion of the liver may be elevated so that a smaller portion of it than is natural is in contact with the abdominal parietes.

In one or more of these ways the normal hepatic dulness may be diminished or may even entirely disappear, so that the pulmonary sound is immediately succeeded by that of the bowel. The liver may thus appear greatly diminished, although its size is not in reality altered. You will find a remarkable case of this sort recorded by Dr. Bright, where, on opening the body, neither the liver nor the colon presented itself to view, but, in their stead, the convolutions of the small intestines, which were found to have come completely in front of the liver, the colon and the omentum doubling over the liver and pressing it back, and having made deep furrows on its anterior surface.[1] The fact of hepatic contraction being of this spurious character ought always to be suspected under the following circumstances :—

a. The very fact of there being tympanitic distension of the bowels ought to suggest caution in inferring the existence of real atrophy of the liver from a diminished area of hepatic dulness. The same caution is necessary in cases of ascites. The fluid in the peritoneum pushes up the bowels which may be only moderately distended with gas, but which may thus come to produce the same result as more extensive tympanites; and this fallacy is of the greater importance inasmuch as ascites is a common consequence of real atrophy of the liver.

b. Variation in the extent of hepatic dulness at different times is a character of spurious atrophy of the liver most useful in diagnosis. The dulness of the liver will vary in its extent according to the amount of gas in the stomach and bowels, or of fluid in the peritoneum. The diagnosis will therefore be facilitated by oft-repeated examinations, and particularly by examinations made before meals, and after the bowels have been cleared out by a purgative.

c. Variation in the extent of hepatic dulness at different places is not uncommon in cases of spurious atrophy. Tympanitic distension of the stomach and bowels may diminish or obliterate the hepatic dulness in the mesial and right mammary lines, but is not likely to affect it materially in the axillary or dorsal lines. Occasionally, too, the space where the hepatic dulness is obscured may be even more circumscribed, as when a knuckle of intestine intervenes between the liver and the abdominal wall.

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[1] Abdom. Tumours, Syd. Soc. Ed. p. 259.

d. The absence of other signs or symptoms of real disease of the liver. The possibility of there being ascites independent of hepatic disease must be kept in view.

2. General or partial *accumulations of gas in the peritoneal cavity*, such as may result from perforation of the stomach or bowels, may obscure, to a greater or less extent, the area of hepatic dulness; but usually the nature of these cases will be sufficiently clear from—

a. The arched tympanitic distension of the abdominal parietes; and

b. Antecedent history of peritonitis from perforation.

3. The hepatic tissue may be *preternaturally soft*, so that the organ may fold on itself and collapse against the spine and the back part of the abdomen, and be covered more or less in front by the stomach and bowels, which may not be abnormally distended with gas. I have already pointed out to you that in fatty degeneration the enlargement may from this cause appear to be increased, a larger portion than natural of the liver being in apposition with the abdominal wall (p. 48); but if the folding be carried a stage further, so as to permit the superposition of bowel, a contrary result may take place. Lastly, in acute atrophy of the liver the organ is not only reduced in size, but it may be so soft as to collapse against the spine, all trace of it disappearing from the abdominal wall in front, so that it may appear smaller than it really is.

Keeping in view these sources of fallacy, which are perhaps more calculated to mislead than the sources of fallacy in the case of enlargement (see p. 7), we may proceed to consider the causes of real atrophy of the liver, which may be conveniently arranged under the three following heads:—

I. Simple Atrophy.

II. Acute Atrophy.

III. Chronic Atrophy; under which head will be included the disease commonly designated 'Cirrhosis,' 'Simple Induration,' and 'Red Atrophy.'

I shall now endeavour to describe to you the leading clinical characters and the appropriate treatment of these several forms of atrophy.

I. SIMPLE ATROPHY.

By 'simple atrophy' is understood a diminution in the size of the liver, independent of any alteration in its structure, except a diminished size of the lobules, which may be so small as to be distinguished with difficulty, the cut surface presenting a smooth appearance and often a uniform tint. The liver in this state may be reduced to less than one-half of its normal weight and bulk. Although this condition of liver is not of much practical importance, more or less of it is far from uncommon, and ignorance of its nature and characters may lead to errors in diagnosis. You will recognise this form of atrophy then by the following characters :—

1. The circumstances under which it occurs. These are mainly two, viz. Old age and Inanition.

a. Old Age—Simple atrophy has been sometimes described as 'senile atrophy.' With the advance of life, the tendency of the various organs and tissues throughout the body is either to degenerate or to waste. In some persons the several forms of *degeneration* (fatty, calcareous, &c.) predominate; while in others we observe a simple *wasting*. In the latter case, the power which prevailed over the waste of the body in childhood and youth, and which maintained the balance in the vigour of manhood, has failed, and waste now prevails over development. Considering the important part played by the liver in the nutrition of the body, it is not surprising that its reduction in old age is in advance of that of the body generally, and occasionally the liver is reduced by senile atrophy to one-half of its normal size and weight.

b. Inanition may also induce simple atrophy. There is little or no supply to compensate for the constant waste. When you remember the increase in the bulk of the liver produced by every meal (see p. 131), you will readily understand how, in cases of inanition, the liver often wastes out of proportion to the rest of the body. It is difficult to say why it is that the effect of wasting disease is in some persons to cause wasting of the liver, while in others it leads to the accumulation in the organ of a large quantity of oil (see p. 51). Inanition may arise in two ways, either from an insufficient supply of food or from diseases which interfere with the assimilation of food. Accordingly you will find simple atrophy of the liver extremely common in the bodies of persons who have died of stricture of the pylorus, or of stricture of the œsophagus or of the cardiac orifice of the stomach. I shall relate to you immediately the particulars of a patient, aged 54, with a cancerous tumour of the lower end of the œsophagus, in whom the area of hepatic dulness was reduced to one half of the normal standard, and whose liver after death was found to weigh only 32 oz., instead of 54 oz., the average weight for his age (Case CI.). You will remember also the case of Samuel H., aged 63, who died of a cancer of the œsophagus involving the apex of the left lung, and whose liver was very small and weighed only 42 oz.; and the case of Eliza P., aged 48, who died of cancer of the pharynx and whose liver weighed only 35 oz. All these were good examples of simple atrophy.

c. External Pressure by tight lacing, pleuritic or pericardial effusions, circumscribed peritoneal exudations, or enlargement of those portions of the bowel nearest to the liver, may likewise produce simple atrophy of the liver. The atrophy, however, under these circumstances is usually partial and is of little clinical importance, unless the bile-ducts or large blood-vessels have been subjected to the pressure.

2. There is an absence of any sign of hepatic disease or derangement. With the diminution in the size of the liver, there is, no doubt, a loss of functional power, but sufficient secreting tissue remains for the diminished work to be done. Care, however, must be taken not to mistake for symptoms of diseased liver those of the primary disease on which the atrophy depends.

Simple atrophy of the liver requires no special *treatment* beyond that adapted to the circumstances under which it occurs.

The following case will serve to impress on your memories the clinical characters and post-mortem appearances of simple atrophy of the liver. The case is also interesting as an illustration of cancerous and tubercular deposit taking place simultaneously, of which other examples have been reported by Mr. Sibley,[1] Dr. Bristowe,[2] and myself.[3] It is difficult to account for these cases on the ordinarily accepted view, that tubercle and cancer depend on a 'peculiar diathesis,' regulating the nature of the exudation, for then the diathesis must vary in different parts of the same body.

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[1] Med. Chir. Trans. vol. xlii. p. 149.
[2] Trans. Path. Soc. vol. x. p. 284.
[3] Ibid. vol. xv. p. 104.

CASE CI.—*Co-existence of Cancerous Stricture of Œsophagus with recent Tubercle in Lungs. Simple Atrophy of Liver.*

Augustus T——, aged 54, a tailor, adm. into Middlesex Hosp. on July 24, 1863. He was of average height, and naturally of spare habit. He had led a very intemperate life, drinking large quantities of gin, but he had always enjoyed good health, until about four weeks before admission, when he began to suffer from sickness, coming on immediately after eating, sometimes even before he thought the food had been swallowed. He had never observed blood in the vomited matters, but he had rapidly lost both flesh and strength.

On admission, very emaciated; an anxious expression of countenance. He could swallow solid food; but it was usually rejected, either immediately or within a few minutes. He also brought up from time to time large quantities of clear acid fluid. He complained of pain between shoulders, but there was no tenderness of spine, and no abnormal physical sign in either lung. Abdomen nowhere tender, and nothing like a tumour could be felt in any part of it. Hepatic dulness much diminished, not exceeding 2½ inches in right mammary line. Splenic dulness normal; no ascites or jaundice. Tongue furred; bowels costive. Pulse 61 and feeble; no abnormal bruit over heart; no anasarca; no albumen in urine.

All remedial measures failed to relieve the vomiting, and patient got rapidly thinner and weaker, while hepatic dulness was reduced to 2 inches. On Aug. 30, vomiting abated, but this was due to patient's taking scarcely any nourishment. He died on Sept 7. At no period of his illness had he cough; lungs not examined after July 24.

Autopsy.—Entire absence of fat beneath integuments and throughout body. Œsophagus, 1½ inch above cardia, had its calibre narrowed to that of a goose-quill for about half-an-inch. A hard tumour, size of half a walnut, was firmly attached to constricted portion, and formed part of its posterior wall; mucous membrane corresponding to this presented a puckered cicatrix-like appearance. Substance of tumour dense, fibrous, white, and slightly translucent, and dotted over with softer, more opaque, yellow specks. It yielded an opaque juice on scraping. On microscopic examination, the firmer portions of tumour were found to contain numerous 'cancer cells,' varying in size up to $\frac{1}{450}$ inch in diameter. They were rounded, elliptical, and caudate, and contained one or two large nuclei with a diameter about one-third of that of cell. Some of cells had smaller cells in their interior. In softer portions of the tumour, cells were ill-defined and mixed with much oily and granular matter. Neither bronchial glands, nor lymphatics in neighbourhood of tumour were enlarged. Stomach small, but otherwise normal. Liver presented ordinary characters of simple atrophy; it weighed only 32 ounces; its outer surface was smooth; only abnormal appearance seen on section was that acini were reduced to one half of their usual size; secreting cells were small, and contained scarcely any oil, but were otherwise normal. Spleen weighed only three ounces; kidneys were also small and anæmic, but in other respects normal.

Both lungs were very small, right weighing 9¾ ounces, and left 8¾ ounces. Apices of both were firmly adherent to thoracic walls, and marked externally with cicatrices. Several cretified deposits as large as peas, as well as one or two small cavities with thick walls and containing pus, were disclosed on cutting into cicatrices. Scattered through upper lobes of both lungs were a number of translucent greyish granules, isolated and collected into groups, as large as a hazelnut, and presenting all the naked-eye and microscopic characters of miliary tubercles. Heart weighed only 6¾ ounces, and was destitute of fat, but in other respects was normal.

II. ACUTE OR YELLOW ATROPHY. 'MALIGNANT,' 'TYPHOID,' OR 'HÆMORRHAGIC JAUNDICE.'

This is a rare but very remarkable disease, in which the liver becomes rapidly atrophied with the development of jaundice and cerebral symptoms, and where after death what remains of the organ is found to be extremely soft and yellow, with no appearance of lobules, and with the secreting cells in a great measure, or wholly, broken up into granular matter and oil-globules. The rarity of the disease in this city is attested by the fact, that although a brown tongue and delirium formerly constituted a certain passport for the transmission of all diseases to the London Fever Hospital, out of about 25,700 cases admitted during nine years, I believe that the only example of the disease which was noticed was one of which I shall narrate to you the particulars immediately. The disease, however, is one of the most interesting that can engage your attention, and may be recognised by the following clinical characters:—

1. Premonitory symptoms are noticed in many cases, but they are usually slight and variable in their nature and they are sometimes absent. The most common are those of gastro-enteric catarrh, such as furred tongue, nausea and loss of appetite, occasional vomiting and irregular bowels—diarrhœa or constipation, and slight pyrexia. At other times, the patient complains only of rheumatic pains, of an uneasy sensation in the region of the heart or stomach, or of a feeling of uneasiness which he is unable to define. These symptoms may last three or four days, or as many weeks, but withal there is

not, as a rule, thought to be much amiss, while in not a few cases the patient has no feeling of indisposition until the supervention of symptoms of a more decided character.

2. Jaundice is invariably present, and is usually the first symptom that attracts attention to the patient's condition. The jaundice, however, is rarely intense, and is sometimes confined to the upper part of the body. Like the jaundice of pyæmia (see p. 165) it appears to be due to a morbid condition of the blood, and is independent of any obstruction of the bile-duct, and bile is found throughout in the stools. (See Lect. XI.)

3. A rapid diminution in the area of hepatic dulness is one of the most remarkable features of the disease. In the course of a week or ten days, one-third, or even more than one-half of the liver may disappear (see fig. 23). Bright has recorded a case where the liver after death weighed only nineteen ounces, and its weight in one case which I shall bring under your notice was only twenty-eight ounces. It must not, however, be inferred that a diminished area of hepatic dulness is necessary for the diagnosis of the disease known as 'acute atrophy.' It may happen that the liver has been enlarged by previous disease (Case CIII.); but independently of this, a considerable number of cases have been observed in which the liver at the commencement of the morbid process, and consequent upon it,

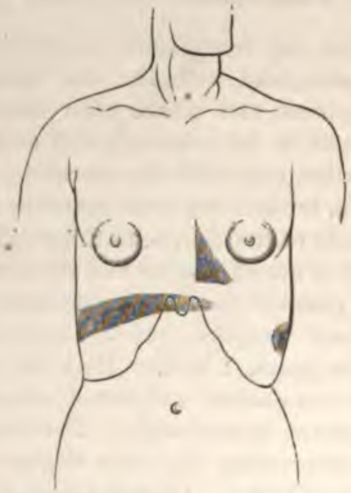

Fig. 23. Area of Hepatic Dulness in Mary Ann M—— (Case CII.), on the day before death.

has been found to be considerably increased in size. Liebermeister[1] and Trousseau[2] refer to cases of this sort; and similar observations have been made by Sieveking,[3] Moxon,[4] Tuckwell,[5] &c. Still, even in these cases, the preliminary enlargement is followed, if the case be sufficiently prolonged, by a rapid diminution in the size of the liver. It has been lately suggested that the atrophy in these cases is a chronic process, though unattended by symptoms until the final explosion; but this view is opposed by most of the known facts in reference to the etiology of the disease, and also by the circumstance that it may be ascertained by percussion that the atrophy is going on during life. Careful examination of the gland after death shows that the atrophy is due to a destructive process commencing at the circumference of the lobules and advancing to the centre, as the result of which the secreting cells disappear and in their place we find nothing but granular matter and oil. The disease, in fact, is believed to be nothing more nor less than an acute fatty degeneration of the liver, resulting from a diffuse inflammatory process; for, previous to bursting, the cells may often be seen distended with oily and granular contents. During life the atrophy of the liver may appear greater than it really is, because the gland is not only reduced in size, but also softened, so that it folds upon itself and collapses towards the vertebral column, the space corresponding to it in front being occupied by intestines containing gas.

4. Pain at the epigastrium and in the region of the liver is present in most cases. This pain often comes on spontaneously, and can almost always be elicited by pressure, even when the patient is almost unconscious. There is rarely, however, any tympanitic distension of the abdomen, unless there be (as in Case CII.) concurrent peritonitis, in which case the pain and tenderness may be acute. Muscular and arthritic pains are also not uncommon and occasionally the joints seem swollen; while some patients complain of suffocative sensations, or there is dyspnœa not accounted for by any lesion in the heart or lungs.

5. Vomiting occurs in most cases, the vomited matters consisting of the ingesta mixed with mucus or bile, but often also containing much blood, and resembling the 'black vomit'

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[1] Virchow's Jahresbericht, 1870, ii. 165.

[2] Clin. Med., Syd. Soc. Ed. iv. 299, 308.

[3] Lancet, 1872, ii. 224.

[4] Path. Trans. vol. xxiii.

[5] Barth. Hosp. Rep. vol. x.

of yellow fever. The bowels are described as being usually constipated, but in the case from which this liver was taken (Case CII.) there was a considerable amount of diarrhœa. The stools in the first instance are often pale, but in other cases they contain bile; in the advanced stage they not unfrequently contain blood and are very offensive.

6. The area of splenic dulness is usually increased, except in cases where the portal system has been drained by diarrhœa or by hæmorrhage from the stomach or bowels.

7. The cerebral symptoms of the 'typhoid state' constitute one of the most frequent and striking peculiarities of acute atrophy. As a rule, they appear simultaneously with the jaundice, but occasionally not for two or three weeks subsequently, the jaundice at first having all the characters of what is commonly known as 'catarrhal.' At first there is headache, with despondency, irritability, and great restlessness; and this condition is succeeded by low muttering delirium, tremors, subsultus, muscular rigidity and carphology, retention or incontinence of urine, involuntary passage of fæces, stupor, coma and convulsions. These symptoms are said to be sometimes associated with fatty disintegration of the cerebral tissue, but, like the analogous symptoms in typhus fever and in the typhoid state generally, to which I have directed your attention on a former occasion,[1] they probably result from the circulation through the brain of blood poisoned by the accumulation in it of urea and other products of tissue-metamorphosis which ought to be eliminated by the kidneys.

8. Acute atrophy of the liver is not attended by pyrexia. The pulse varies. In cases ushered in with gastro-enteric catarrh the pulse is usually accelerated at first, but falls to the normal standard, or below this, on the appearance of jaundice, and again rises on the supervention of cerebral symptoms, its frequency often varying at different hours of the day. In Case CII. it rose to as high as 144, but here there was peritonitis. The temperature in the early stage of the disease may be slightly elevated, but it rarely much exceeds 101°. But when the symptoms are well pronounced, the temperature is usually not elevated, and sometimes it is subnormal. In Case CIII. it fell to as low as 95·5° and a similar observation has been made

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[1] On the Pathology and Treatment of the Typhoid State in different Diseases. Abstract of Lecture in Brit. Med. Journ. Jan. 4, 1868.

by Duckworth.[1] The temperature of 101° noted shortly before death in Case CII. was probably due to the peritonitis. This absence of pyrexia is of the utmost importance in the diagnosis of acute atrophy from other diseases characterised by the typhoid state. After the appearance of cerebral symptoms, the tongue is almost invariably dry and brown, and the teeth crusted with sordes, exactly as in a bad case of typhus fever.

9. The urine undergoes important changes. Its quantity is not materially altered; it is of acid reaction; and its specific gravity varies from 1012 to 1024. Its colour is usually dark, but the ordinary reaction of bile-pigment may be faint or indistinct. It often contains albumen or even blood; but after the removal of the urinary pigment, it yields no reaction of bile-acids to Pettenkofer's test. The most remarkable alterations, however, consist in the great diminution or even total disappearance of the urea and uric acid, and also of the chlorides, sulphates, and earthy phosphates, and the substitution of two new substances of a peculiar nature, leucin and tyrosin. These substances are products of the metamorphosis of nitrogenous matter intermediate between the protein principles (albumen and fibrin) at one extreme, and the less complex bodies, urea, uric acid, kreatin, &c. at the other, as will be seen by a comparison of the following formulæ:—

Albuminoids = $C_{72}H_{112}N_{18}SO_{23}$
Tyrosin = $C_9H_{11}NO_3$
Leucin = $C_6H_{13}NO_2$
Uric Acid = $C_5H_4N_4O_3$
Urea = CH_4N_2O

Leucin and tyrosin, in the crystalline forms represented in the annexed figures (figs. 24, 25, and 26), are found in the tissues of the liver, spleen, and kidneys in cases of acute atrophy, and they are usually also secreted in large quantity in the urine, from which they separate as a distinct deposit on standing, or they may be obtained by evaporating a few drops of the urine, on a glass slide.[2] The detection of these crystalline bodies in the urine of a case of jaundice may be said to clench the diagnosis of acute atrophy of the liver, but the failure to detect them must not exclude acute atrophy from the diagnosis. For instance, they were not present in the urine of Case CII., at all events in such quantity as to reveal their existence by simply evaporating the urine, although they were found in considerable quantity in the liver and kidneys after death. It is true that in this case death was accelerated by acute peritonitis; had the patient survived a little longer, leucin and tyrosin would probably have been found in the urine.

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[1] Barth. Hosp. Rep. vol. vii. Bright and Alison long ago noted that the skin was cool.

[2] Tests for Leucin and Tyrosin. A. For Leucin. Concentrate urine and dissolve in alcohol. Evaporate alcoholic solution and dissolve in water, from which the leucin ought to crystallize in spherical globes. B. For Tyrosin. 1. *Hoffmann's Test.* A solution of the nitrate of the protoxide of mercury, nearly neutral, is to be added to the suspected solution. If tyrosin be present, a reddish precipitate is produced, and

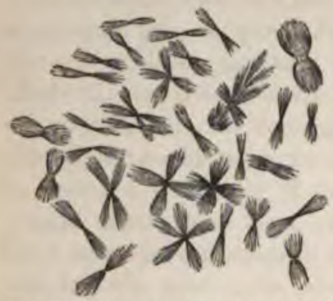

Fig. 24. Microscopic needle-shaped crystals of tyrosin adhering in bundles and in stellate groups.

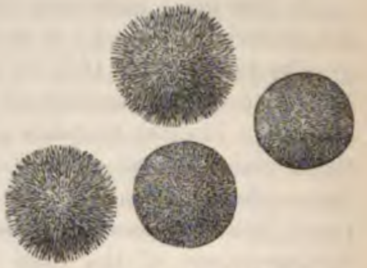

Fig. 25. Microscopic globular masses composed of acicular crystals of tyrosin.

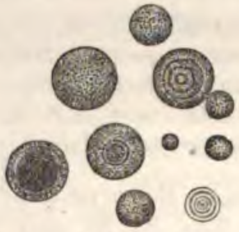

Fig. 26. Microscopic laminated crystalline masses of leucin.

10. Hæmorrhages are very common, and particularly hæmorrhage from the stomach, bowels, or nose. Blood is often vomited in large quantity. Petechiæ, purpura-spots, and vibices often appear on the skin, or in rarer cases there is uterine hæmorrhage. After death ecchymoses are found in different parts of the body. From the frequency of these hæmorrhages

the supernatant liquid is of a dark rose colour. 2. *Frerichs' Test.* Add to suspected liquid a solution of acetate of lead until no more precipitate is produced. Sulphuretted hydrogen gas is then to be passed through the filtered fluid. Separate the sulphuret of lead by filtration, and concentrate the clear solution by evaporation, when, if tyrosin be present, it will crystallise out as long white needles.

the disease has been sometimes designated 'hæmorrhagic jaundice.'

11. Pregnant females, who constitute a large proportion of the cases, almost invariably abort or miscarry before dying.

12. The circumstances under which acute atrophy of the liver occurs constitute not the least interesting part of its clinical history. The causes of the disease still require investigation, but I shall briefly mention those that are at present known. Among *predisposing* causes, then, we have—

a. Age. Most persons attacked with the disease are under the middle age. Of 31 cases collected by Frerichs, 26 were under 30, and all but 2 under 40. Of 4 cases which have come under my notice, two were females, aged 19 and 30; and two were males, aged 24 and 62; in a fifth case (Case CIII.), where the disease was secondary to obstruction of the bile-duct, the patient was a male, aged 66. All writers agree as to the rarity of the disease in children. Neither Niemeyer, Frerichs, nor Trousseau ever met with the disease in early life. West, in his extensive experience, has only seen the disease once, in a girl aged 4½ years; but three other cases in children have been recorded by Duckworth[1] and Tuckwell.[2]

b. Sex. The disease is much more common in females than in males. Of the 31 cases collected by Frerichs, 22 were females.

c. Pregnancy must be regarded as a predisposing cause, for of the 22 female patients referred to by Frerichs, one half were attacked while pregnant. From the third to the sixth month is the most common period of pregnancy at which the disease shows itself. In the pregnant state it is said to be frequently associated with fatty degeneration of the kidneys and albuminous urine.

d. Dissipation, including drunkenness and venereal excesses, precedes the disease in a considerable number of cases. Leudet is of opinion that its origin may sometimes be traced to the absorption of a large quantity of undiluted alcohol.[3]

e. Constitutional syphilis appears to be a predisposing cause in some cases. Most writers on syphilis have noted the frequent occurrence of jaundice about the commencement of what is known as the secondary stage; in most cases the constitutional symptoms are slight and the jaundice soon passes away (p. 153); but now and then what appears to be at first a slight catarrhal jaundice becomes rapidly developed into the symptoms of acute atrophy.[1]

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[1] Loc. cit.

[2] Loc. cit.

[3] Clin. Médicale, Paris, 1874, p. 67.

Among causes that appear to act more directly in *exciting* the disease are the following:—

a. Nervous influences, such as severe mental emotions, and particularly anxiety, fear, and grief. Sir Thomas Watson, in his lectures, states that scores of instances are on record, where jaundice has suddenly appeared under such circumstances, and adds that 'these cases are often fatal, with head symptoms, convulsions, delirium, or coma, supervening upon the jaundice.'[2] In these cases an impression made upon the nervous system may be directed to the liver and derange its nutrition, or perhaps more probably it excites in the first instance morbid changes in the blood.

b. Malaria. There are other cases where the disease has apparently resulted from some malarious poison, acting probably through the blood and the nervous system. Instances have been recorded by Graves,[3] Budd,[4] and others, where several cases of what appears to have been unquestionably this disease occurred in the same house, or where it has been even epidemic in certain localities (See Lecture XI.); and when it is considered what a rare disease acute atrophy is, it is impossible to escape from the conclusion that in these cases there must have been some local cause to which all the patients were subjected in common.

c. The blood-poisons of typhus fever and allied diseases have been known to give rise to acute atrophy of the liver.[5] Jaundice is a very rare complication of typhus and scarlet fever, but in more than one instance where it has occurred,[6] I have found crystals of leucin and tyrosin in the tissue of the liver and kidney. The liver in these cases has been in a state of fatty degeneration, but without marked atrophy. Most writers on the yellow fever of the tropics have described fatty degeneration of the liver as one of its most characteristic lesions, and it is well known that there is a deficient elimination of urea; but observations are still wanting as to the presence or absence of leucin or tyrosin in the urine and in the tissues of the kidneys and liver.

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[1] See Lebert in Virchow's Archiv, 1854, 1855; Andrew, in Path. Trans. xvii. p. 158; and Fagge, Ib. xviii. p. 138.

[2] Lectures on the Practice of Physic, 5th ed. ii. p. 682.

[3] Clin. Lect. 2nd ed. ii. p. 255.

[4] Op. cit. 3rd ed. pp. 255, 270.

[5] See Frerichs' Treatise on Diseases of Liver, Syd. Soc. Ed. i. p. 235.

[6] Treatise on the Continued Fevers of Great Britain, 2nd ed. p. 210.

d. Phosphorus occasionally produces symptoms and structural changes in the liver very similar to, if not identical with, those of acute atrophy of the liver. (See Lect. XI.)

e. Lastly, it seems not improbable that in some cases of acute atrophy the cause may be, as suggested by Dr. Budd[1] and Trousseau,[2] some special poison engendered in the body itself by faulty digestion or assimilation. The nervous influences already referred to may possibly contribute to the development of such a poison. It is probably also in this way that acute atrophy sometimes supervenes upon other diseases of the liver. According to Trousseau, malignant jaundice is 'never caused by obstruction of the biliary ducts;'[3] but this assertion is negatived by what was observed in Case CIII. During a prevalence of epidemic catarrhal jaundice it has frequently been found, that while the majority of the patients have recovered without any bad symptoms, a few, at first in no way to be distinguished, have been followed by acute atrophy. A similar observation has been made in the catarrhal jaundice resulting from syphilis.

It would, indeed, be remarkable that all these causes should single out the liver for special destruction; but in a future lecture I will show you that the liver is far from being the only organ that undergoes disintegration, and that in fact the disease known as 'acute atrophy of the liver' is more probably a general than a local disease. (See Lect. XI.)

Treatment.—In acute atrophy of the liver all treatment has hitherto proved unsatisfactory. The disease, after the supervention of cerebral symptoms, is in most cases fatal, although well authenticated instances are on record where patients have recovered after falling into a state bordering on coma. It may be well, therefore, to enumerate those remedial measures which have appeared most useful, or which seem indicated by our knowledge of the pathology of the disease.

1. Purgatives. In several instances which have been reported as occurring in Ireland, patients in the same house with others who have died have recovered after active purging in

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[1] Op. cit. p. 265. [2] Op. cit. p. 319. [3] Op. cit. p. 317.

conjunction with leeches and blisters to the head.[1] Even in fatal cases temporary improvement has often followed smart purgation. Dr. Budd also states that in several cases he had found advantage from a combination of sulphate of magnesia (ʒj), carbonate of magnesia (gr. xv), and spiritus ammoniæ aromaticus (ʒss) given three times a day.

2. After the supervention of cerebral symptoms, all measures calculated to promote the elimination of urea and uric acid or of other products of disintegrated tissue from the system deserve a trial. It is in this way perhaps that purgatives have proved beneficial, and that warm baths, hot air baths, diaphoretics, diuretics, and colchicum may also be expected to do good.

3. In cases where there is extensive hæmorrhage from the stomach or from other mucous membranes, ice or astringents may be necessary.

4. It is in the early stages, however, of the malady, before the occurrence of cerebral symptoms, that most advantage may be expected from treatment. Cheering society, holding out hopes of recovery, change of scene, anodynes to procure sound sleep, attention to the condition of the stomach and bowels, and ammonia and alkalies with the infusion of gentian or some other vegetable bitter, are the measures which appear best calculated to avert those terrible cerebral symptoms from which so few recover.

The liver, which I show you here, was taken from the body of a patient who died some years ago in the London Fever Hospital, and who presented the symptoms of acute atrophy of the liver in a typical form, excepting that no leucin or tyrosin was found in the urine passed the day before death. These substances, however, were detected after death in the tissue of the liver and kidneys, and the former of these organs presented all the anatomical characters peculiar to the disease. It may be worth mentioning, however, that both Dr. Cayley and myself failed to find either leucin or tyrosin in the fresh liver and kidneys, although they were present in large quantity after these organs had been immersed for some days in spirit.

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[1] See cases by Dr. W. Griffin, of Limerick, in Dub. Journ. of Med. and Chem. Science, 1834, and by Dr. Hanlon, in Graves, *loc. cit.*

CASE CII.—*Acute Atrophy of Liver—Acute Peritonitis—Leucin and Tyrosin in Liver and Kidneys, but none detected in Urine.*

Mary Ann M——, a sempstress, aged 19, admitted into London Fever Hospital, on evening of Feb. 13, 1868, and seen by me on following morning. She was unmarried. Her father was a German, but she had been born and brought up in London. Her sister was not aware that she had suffered from any mental trouble, and believed that her catamenia had been regular; there was no history of syphilis. There had been no other case of illness in the house from which she came. She had been quite well until middle of January, when she began to complain of loss of appetite and nausea, and after ten days, her skin was noticed to be slightly yellow. A week before admission she took to bed, complaining of pain in region of stomach, aggravated by any movement, but unattended by vomiting. For about a fortnight before admission bowels had been relaxed three or four times a day, motions at first being yellow, but latterly green. Three days before admission she began to be 'light-headed.'

On morning after admission the following note was taken:—'Patient is a well-nourished girl, and has deep jaundice of skin and conjunctivæ. Is scarcely conscious, and can give no account of herself. Since admission has been very restless and delirious, often screaming out loudly. Pupils much dilated, but equal. No eruption on skin, which feels dry and hot, temperature in the axilla being 101° F. Pulse 116 and weak. Cardiac and respiratory signs normal. Tongue dry and brown, and since admission there has been frequent vomiting of a dark brownish fluid evidently containing blood. Bowels have acted several times, and from nurse's account, who describes motions as very dark, watery, and offensive, they have probably contained blood. Abdomen moderately distended and tympanitic; pressure upon it does not seem to cause pain, but respiration is thoracic, and there is an obscure thrill, as from fluid, on tapping both flanks. Hepatic dulness is greatly diminished, not exceeding 1¼ inch in right mammary line, and its lower margin being fully 2 inches above that of ribs (se fig. 23, p. 260). Urine has been passed in bed, but bladder is now full.'

About two pints of urine were drawn off by catheter, which had the following characters. It was acid; specific gravity 1015; of a dark greenish-brown colour, but presenting reaction of bile-pigment in only a faint degree. Heat produced no change on it, but on adding nitric acid, after boiling, it became turbid, as well as very dark. Nitrate of urea could be obtained from it in only very small quantity, but no crystals of leucin or tyrosin could be detected, either as a separate deposit on standing, or after evaporation of a few drops of urine in a watch-glass. Unfortunately urine was thrown away before it could be submitted to a more careful analysis.

Patient was ordered a mixture containing nitric acid, nitrous ether, and nitrate of potash, with milk, beef-tea, and four ounces of gin. She became rapidly worse, although she was less noisy and delirious, and seemed to sleep a good deal at intervals. Diarrhœa continued, motions being passed in bed, and being still liquid and very offensive, but of a light yellow colour. In evening of 14th, pulse 144; respiration 32 and thoracic; temperature in axilla 100·8°. She continued much in same state, and died, without any convulsions, at 7.50 a.m. on following morning, five days after first appearance of cerebral symptoms.

Autopsy.—Body well nourished. Much purple lividity of integuments, and deep jaundiced hue of skin and of every tissue of body. No scars on genitals or in groins. Three or four pints of slightly turbid serum in peritoneum. Considerable fine vascular injection of serous covering of small intestines, and particularly of that of duodenum; peritoneum of intestines and of liver also coated at many places with a thin film of recent lymph, easily separated. Stomach and intestines distended with gas, and liver completely hidden below right ribs, not more than an inch of it being opposed to thoracic wall. Liver extremely small; its largest diameter measuring 6½ inches, and antero-post. diameter of right lobe only 5 in.; it weighed only 28 ounces, or exactly one half of standard weight for girl's age; very flabby, and outer surface wrinkled, but free from any granular or nodular irregularities. The substance of gland extremely friable, and of almost pulpy consistence, and presented at some places a tolerably uniform rhubarb-yellow colour, with scarcely any appearance of lobules, and at other parts a similar yellow colour interspersed with red. Under microscope, there was found a large quantity of free oily and granular matter, with globular masses of leucin and bundles of needles of tyrosin, and also, more especially at what corresponded to centres of lobules, entire secreting cells of large size and loaded with oil-globules and dark greenish-yellow pigment. Bile-ducts patent throughout, not dilated; their lining membrane presented no tinge of bile, although gall-bladder contained about a teaspoonful of dark-green viscid bile, which could be squeezed out through cystic duct. Contents of intestine consisted throughout of a very pale yellowish pulp; mucous membrane of bowels nowhere ulcerated. Spleen of normal size, rather soft. Both kidneys slightly enlarged, extremely soft, and tinged with bile pigment; renal epithelium contained a large quantity of fine granular matter; crystals of both leucin and tyrosin were detected in renal tissue. Bladder empty and uterus unimpregnated. Much hypostatic congestion of both lungs. Pericardium contained more than an ounce of yellow serum; heart healthy; blood dark and fluid. Excepting an increased amount of serosity in lateral ventricles and beneath arachnoid, neither brain nor membranes presented anything abnormal.

Case CIII.—*Jaundice from Gall-stones, followed by Acute Atrophy of the Liver, with Puriform Deposits.*

James H——, æt. 66, was adm. into Middlesex Hosp. on Oct. 11, 1870. As a tailor he had led a sedentary life; he had lived well and drunk a good deal of beer, but at no time been intemperate. Excepting slight cough and occasional symptoms of indigestion, and three attacks of gout in big toe, he had enjoyed good health until seven weeks before admission, when he was suddenly seized in night with violent pain in epigastrium and right hypochondrium, frequent vomiting, rigors, and cold perspirations. On following day he was jaundiced. The jaundice disappeared after a few days; but the pain and sickness continued to recur at frequent intervals, and a week before admission he had a severe attack, followed by jaundice, which persisted.

On admission, jaundice was patient's prominent symptom; much bile-pigment in urine, but none in fæces. Liver enlarged, measuring 5½ in. in right nipple line. Excepting the jaundice, patient's general aspect was that of a healthy man for his age. Pulse 60; skin cool; tongue coated; solid food was at once rejected by vomiting; bowels open by medicine. No albumen in urine. On following day, *Oct.* 12, patient's condition was entirely changed. Pyrexia had set in; pulse 96, and temperature 101·2° Expression heavy and stupid. No pain complained of. *Oct.* 14.—Tongue dry and brown down centre. Jaundice and vomiting persisted. No rigors or perspirations. *Oct.* 16.—Tongue dry all over; much thirst; bowels not open; frequent hiccough; drowsy and heavy, but did not wander. Pulse 92; temp. 100·6°; no rigors nor perspirations. *Oct.* 17.—Pulse 68; temp. 97·2° in morning, and 101·6° in evening. Urine contained much bile-pigment, and also crystals of tyrosin and leucin, but no albumen. *Oct.* 18.—Pulse 84 to 120; temperature 100·1° to 103·1°. Much hiccough. Urine passed involuntarily. *Oct.* 19.—Stupor increased. Urine still contained tyrosin, but no albumen. Liver appeared to be diminishing in size, and did not exceed 4 in. in right nipple line. *Oct.* 22.—Pulse 80 in the morning, 140 in evening; temp. 96·6° in morning, 102·2° in evening. Less hiccough; abdomen distended and tympanitic. Still much jaundice, but motions now contained bile. All day he lay in a heavy drowsy state, but in evening he became very restless, tossing about and throwing off bed-clothes. Hands tremulous, occasional delirium. *Oct.* 26.—Pulse 100; temp. 101·2°. Urine still contained tyrosin and fair amount of urea, but no albumen. *Oct.* 27.—Pulse 96; temp. 98·4° to 100°. Considerable delirium, and frequent attempts last night to get out of bed. Still no rigors nor sweating. Urine contained a trace of albumen. *Oct.* 28.—Pulse 128; temp. 103·2° *Nov.* 2.—Pulse 80; temp. 96·8°; last night it was as low as 95·5°, and at no time during last two days has it exceeded 98·2°. Patient still very restless, but much weaker; now almost unconscious, with occasional muttering delirium. Hepatic dulness in right nipple only 3½ in. Jaundice decidedly less; bile in motions. Urine copious; it contained very little bile-pigment, but a good deal of tyrosin, and about one-twelfth in volume of albumen. *Nov.* 3.—Pulse felt with difficulty; temp. 98·6°. *Nov.* 4.—Pulse scarcely to be felt; temp. 96·6°; voice feeble, and could not speak articulately; quite unconscious, restless and moaning; tongue dry and brown. Urine scanty; contains one-twelfth in volume of albumen and much leucin and tyrosin, but very little urea; less jaundice; complexion dusky; no purpuric spots. In early part of following night acute delirium set in, followed after some hours by great restlessness. At 5 a.m. of Nov. 5th he became quiet, but his respirations were quick (48). At 11.30 a.m. death occurred, being preceded by slight convulsions.

Autopsy.—Body thin; only faint jaundice of skin and tissues; no purpura-spots. No peritonitis, old or recent. Surface of liver smooth; capsule not thickened. Liver large and heavy, 94 oz., but not so much as three inches apposed to wall of chest and abdomen, the organ being soft and folded upon itself, and overlapped to an unusual extent by right lung. On cutting into liver it presented numerous patches of yellow opaque fluid having all naked-eye characters of pus contained in cavities with well-defined walls, which were apparently dilated bile-ducts. These cavities varied from size of a pea to that of a small cherry. Under microscope the yellow fluid was found to contain a few pus-corpuscles, but to be made up chiefly of oily matter. The hepatic tissue was unusually soft, and of a yellowish or rhubarb colour. It contained many tracts where it was impossible to trace any outline of lobules, and where the liver-cells were replaced by oil, granular-matter, and round nuclei. No leucin nor tyrosin could be found at first, but after liver had been for some time in spirit it was found to contain many crystals of tyrosin. The gall-bladder contained more than twenty polyhedral calculi, about the size of peas. The cystic duct was so dilated that the little finger could be inserted into it. The hepatic and common ducts were also much dilated; the tip of index finger could be passed into either of them. The duodenum for three or four lines round orifice of common duct was ulcerated; it contained bile, which could also be squeezed into it from the gall-bladder. All the ducts in interior of liver were dilated, and the hepatic duct contained three calculi larger than those in gall-bladder, each being about half size of a cherry. No gall-stones were found in bowels. Spleen large and soft, 7¾ oz. The kidneys were congested; each weighed 6½ oz.; both were marked by old cicatrix-like depressions on surface, and there were several small cysts in cortex of the right; both kidneys also contained a few minute opaque yellow soft masses of pus; in other respects they appeared healthy. Heart 14 oz.; valves healthy. Lungs congested posteriorly, but otherwise healthy.

This patient's symptoms left no doubt that his illness commenced with the passage of gall-stones; but it was equally clear that there was some cause other than gall-stones for jaundice which persisted long after the motions contained bile, and which was accompanied by pyrexia and by the cerebral and other symptoms of the typhoid state.

Jaundice with fever and cerebral symptoms, and with bile in the motions, is due to one of three causes:—

1. A specific poison, such as that of yellow fever, relapsing fever, or typhus. 2. Pyæmic abscesses of the liver. 3. Acute atrophy of the liver. With regard to the first cause there was no evidence that the patient was suffering from any of the acute specific diseases, so that the question to be decided was whether he had acute atrophy or pyæmic abscesses of the liver. The latter view was favoured by—*a*, the large size of the liver, and *b*, the fact that gall-stones are known to cause ulceration of the biliary passages, with secondary pyæmic inflammation of the liver. It was contraindicated, however, by—*a*, the absence of rigors or profuse perspiration throughout the entire illness, although both these symptoms are sometimes absent in pyæmia from internal causes; and *b*, the fact that the liver diminished in size, instead of increasing, as the disease advanced. Acute atrophy of the liver was contraindicated by—*a*, the large size of the liver, and *b*, by the comparatively chronic course of the malady; but two facts were strongly in favour of it, viz., *a*, the circumstance that the liver diminished in size as the disease advanced; and *b*, the presence of leucin and tyrosin and the diminution of the urea in the urine. The diagnosis arrived at was that the liver had become enlarged from obstruction of the bile-ducts by gall-stones, and that atrophy of the liver had supervened on this, and had continued after removal of the obstruction. Such cases are referred to by Frerichs in the following passage of his work on 'Diseases of the Liver.'

'In some cases when obstruction of the bile-duct has lasted for several months, it gives rise to an atrophy of the gland, which in many points resembles acute atrophy. The organ diminishes in size and becomes soft; the cells of the parenchyma, which are infiltrated with bile, become disintegrated into a finely granular débris, mingled with oil-globules and particles of pigment, while at the same time large quantities of leucin and tyrosin may be detected.'[1]

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[1] Syd. Soc. Transl. vol. i. p. 237.

In the case now recorded it is to be noted that the process of atrophy continued after the removal of the obstruction.

The autopsy in this case, however, disclosed not only atrophy of the hepatic tissue, but puriform collections in the liver. It is true that these collections were composed mainly of oily matter, yet the ulcerations at the duodenal orifice of the common duct, the great variations in the temperature observed during life, and the fact of a few minute deposits of pus being found in the kidneys, all suggested that the patient was the subject of pyæmic inflammation, as well as of acute atrophy of the liver.

III. CHRONIC ATROPHY.

Under this head it will be convenient to consider several diseases, which in their etiology and anatomical characters are essentially distinct, but which often present symptoms so similar that it may be impossible during life to distinguish them. The diseases I refer to are these:—

I. Cirrhosis, or the so-called 'gin-drinker's liver,' in which the liver becomes reduced in size in consequence of an atrophy or slow destruction of the secreting tissue, but where the fibrous tissue is increased in amount, so that the organ is preternaturally dense and firm. The outer surface also presents a granular or nodulated character, which has earned for the disease the designation of 'hobnailed liver,' and on section the organ presents firm fibrous bands, including the remains of vessels and bile-ducts and surrounding islets of yellow[1] secreting tissue. The capsule also is sometimes thickened and adherent to surrounding parts. In a former lecture I have pointed out to you that the contracted state is very often preceded by considerable enlargement of the liver. True cirrhosis can almost invariably be traced to the abuse of strong spirits, and especially to the habit of drinking them neat, and accordingly it is most common in those countries and towns where such a habit prevails. (Case CXI.).

II. Hyperæmia from obstructed circulation in cardiac and pulmonary diseases causes, in the first place, enlargement of the liver (see pp. 133, 137) ; but when of long standing, the enlargement is followed by an opposite condition of atrophy. The liver also becomes firm, tenacious, and finely granular, and presents an appearance which has frequently been mistaken for cirrhosis. The depressions, however, correspond to the centre of the lobules, whereas in true cirrhosis they are at the circumference. The atrophy is due to the pressure exerted by the distended capillaries of the hepatic vein on the surrounding secreting cells. These cells disappear, so that the central portions of the lobules sink down, while the portions occupied by the branches of the portal vein project as fine granulations. After a time the atrophy extends to the circumference of the large branches of the hepatic vein, so as to cause extensive depressions, and new connective tissue is developed around the vessels, imparting to the organ a greater degree of firmness, and more or less obstructing or obliterating the minute branches of the portal vein. This condition of liver is not uncommon in cases of valvular disease of the heart of long standing. (Case CXIV.).

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[1] This yellow colour is due to the large quantity of yellow pigment contained in the secreting cells. It is from this character that the term cirrhosis (*κιῤῥός*, yellow), is derived, and the application of the term to diseases of other organs, such as the lungs, or kidneys, which resemble cirrhosis of the liver, not in the yellow colour, but in the fibroid condensation of the tissue, is obviously inappropriate.

III. An atrophy of the liver where the organ also presents a granular or nodulated outer surface resembling what is seen in true cirrhosis, but where the fibrous tissue is not increased, so that the liver instead of being preternaturally dense is softer than in health. In some, if not all, of these cases there is no history of spirit-drinking (see Case CXII.)

IV. An atrophy of the liver resulting from frequent attacks of peri-hepatitis or inflammation of the capsule. In these cases the capsule becomes greatly thickened and is often connected to surrounding parts by firm bands of adhesion. Fibrous bands also pass from the thickened capsule into the interior of the liver, which on section often presents a dense, smooth, uniform surface, with the outline of the lobules more or less obliterated. This condition of liver has been described by some writers under the name of 'simple induration,' and is most common in patients who have suffered from constitutional syphilis; it is also met with occasionally in cases of valvular disease of the heart of long standing, in ague, and in connection with inflammation of the right pleura, ulceration of the mucous membrane of the stomach, and various diseases of the secreting tissue of the liver itself. In these cases inflammation is propagated to the capsule of the liver through the diaphragm, along the coronary ligament, or from the subjacent glandular tissue. When the disease has a syphilitic origin, the surface of the contracted liver is often marked by cicatrix-like depressions or deep fissures, giving the organ a lobular character, and gummatous tumours are found in the interior; under other circumstances, the outer surface is smooth, and it never presents the hobnailed character of true cirrhosis. Now and then the fibroid tissue developed in the portal fissure from the products of inflammation produces constrictions of the bile-duct or portal vein.

V. In the next place there is the 'chronic atrophy' of Frerichs, or the 'red atrophy' of Rokitansky. Here there is no nodulation or granulation of the outer surface, and not necessarily any thickening or adhesions of the capsule, but the secreting tissue contains a large quantity of blood, and presents on section a dark-brown or bluish-red colour, a rather firm consistence, and a homogeneous appearance with little or no indication of a division into lobules. The secreting cells are often smaller than natural, and loaded with brown pigment-granules. The atrophy of the organ is general, although its thickness often preponderates over the other dimensions, and occasionally there is a broad rim of atrophied hepatic tissue round the edge. The entire organ has been known to weigh only 24 oz. But the most important anatomical character is the destruction of the ramifications of the portal vein, the branches of which terminate in blind club-shaped extremities, so that the organ cannot be minutely injected from the portal vein. This form of atrophy is occasionally seen in connection with simple and cancerous ulcerations of the stomach and intestines, or in the bodies of persons who have suffered long or often from intermittent or remittent fevers: in the latter case there is often a deposit of black pigment in the minute vessels of the liver.

In all of these diseases there is one anatomical character in common, viz.—a destruction to a greater or less extent of the minute branches of the portal vein in the interior of the liver. To this cause must be attributed the clinical symptoms in which during life they so closely resemble one another. The prominent symptoms in all of them are those of obstructed portal circulation. It will be convenient therefore to describe to you in the first place the typical symptoms in a case of true cirrhosis, and afterwards to mention those circumstances which may serve to distinguish from it the other forms of chronic atrophy.

I. First then let us consider the clinical characters of true cirrhosis. This is a chronic disease; its history usually extends over several years, and may be conveniently divided into two stages, that which precedes, and that which follows, the destruction of the minute branches of the portal vein.

A. *First Stage.*—The disease at its outset is usually insidious.

1. The early symptoms are those of alcoholic dyspepsia, such as retching in the morning and a feeling of sinking inducing a craving for alcohol, loss of appetite for solid food, furred tongue, bitter taste, flatulence and pain after food, attacks of diarrhœa alternating with constipation, hæmorrhoids, urine dark and frequently turbid from lithates and sometimes containing bile-pigment, and languor with depression of spirits. The intensity of these symptoms varies at different times, and at intervals the patient may seem perfectly well.

2. After a time the patient becomes thin and sallow, and venous stigmata are developed on the cheeks. A dull pain with slight tenderness in the right hypochondrium is often present, and pain is sometimes referred to the right shoulder.

3. Your attention has been already called to the circumstance that with these symptoms the liver is often considerably enlarged (see p. 139).

4. The disease also commences now and then in a more acute manner, with febrile symptoms, pain in the hepatic region, vomiting, jaundice, and diarrhœa. But in these cases there has probably been chronic mischief previously, and the acute symptoms have followed some imprudence in diet or unusual excess in stimulants, or a chill. (See Appendix.)

5. There is a history of an immoderate use of wine or spirits, and particularly of the habit known as 'nipping.' The patient rarely takes sufficient stimulants to affect the brain, and is often indignant at the suggestion that he has exceeded a moderate allowance, but you will remember what I told you in a former lecture (p. 141), that what is a moderate allowance for one person may excite serious disease in another.

B. *Second Stage.*—The symptoms in this stage are usually well marked, and are mainly due to the obstructed portal circulation.

1. The area of hepatic dulness is diminished (see fig. 27). It may be reduced to one-half of the normal standard, or even to less. The atrophy is usually greatest in the left lobe, the dulness of which may entirely disappear. The dulness also of

the right lobe may be reduced to a greater extent than would be accounted for by the actual decrease of the liver, owing to its lower edge being tilted up by the pressure of fluid in the peritoneum, or of gas in the bowels, which at the same time increases the antero-posterior diameter of the abdomen, and diminishes the extent of liver which is in apposition with the abdominal parietes. It must not be forgotten, however, that the interstitial hepatitis which leads to cirrhotic contraction may induce all the signs of portal obstruction, although the liver is considerably enlarged (see pp. 139, 142).

2. The nodulated or hobnailed character of the outer surface of the liver may sometimes be felt through the abdominal parietes, and lend assistance to the diagnosis. More commonly, by the time that the irregularity of the surface is sufficient for this purpose, the organ is so small that its lower margin is concealed by the ribs, or by fluid in the peritoneum. In those cases where the liver is enlarged (see pp. 32, 139) its outer surface may be marked by large nodules, separated by deep fissures, and this nodulated character may be easily distinguished through the abdominal parietes, and, as I have already shown you (p. 213), may simulate cancer. In regard to diagnosis also, it is necessary to keep in view the possibility

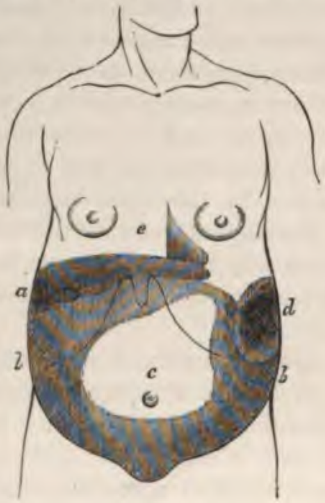

Fig. 27. Shows the hepatic and ascitic dulness in cirrhosis. Thomas B——, Case CIV. (p. 291).

a. Dulness of contracted liver. *b*. Fluid in peritoneum causing bulging of flanks. *c*. Tympanitic bowels. *d*. Enlarged spleen. *e*. Heart.

of a nodulated character of the liver being congenital, or the result of obliteration of large branches of the portal vein. In the latter case, the surface of the liver presents deep fissures, caused by the atrophy of the glandular tissue, which had formerly been supplied with blood by the obliterated vessel.

3. Ascites. A dropsical collection of fluid in the peritoneum, without any pain or tenderness of the abdomen, is one of the most common results of portal obstruction, and is met with oftener in cirrhosis than in any other disease of the liver. The fluid in the peritoneum is a clear yellow serum, having a specific gravity of from 1012 to 1016, containing a large quantity of albumen, but no blood or inflammatory products. In consequence of the distension of the veins which return the blood from the peritoneum, the serous portion of the blood transudes through the walls of the vessels into the peritoneal cavity. When once it appears, it persists and gradually increases. When the amount of fluid is large, it may compress the inferior vena cava and the iliac veins, and thus produce secondary œdema of the legs: but it is a peculiarity of dropsy from uncomplicated portal obstruction, that the ascites precedes and preponderates over dropsy elsewhere. A great amount of ascites may also interfere with the action of the diaphragm and cause much embarrassment of the breathing, but it is distinguished from the ascites produced by cardiac disease, by the fact that the dyspnœa follows and never precedes the ascites.

Frerichs noted ascites in only twenty-four out of thirty-six cases of cirrhosis: patients with cirrhosis may no doubt die in various ways before there is any ascites, but in the advanced stages of the disease ascites is rarely absent.

4. Enlargement of the spleen causing an increased area of splenic dulness is another common consequence of the mechanical obstruction to the circulation through the liver, although, on the whole, it is less common than might at first be imagined. It is present in about one-half of the cases. Its absence is sometimes attributable to fibrous thickening or calcification of the capsule interfering with the dilatation of its contained vessels, and at other times to an excessive drain from the gastro-intestinal mucous membrane, caused by diarrhœa or hæmorrhage.

5. Enlargement of the superficial veins of the abdomen, especially on the right side and between the sternum and the

umbilicus, is another result of the impediment to the current of portal blood, usually observed in advanced cirrhosis. When the portal vein is obstructed, in consequence of the anastomoses between the inferior mesenteric and the hypogastric veins through the inferior hæmorrhoidal, the blood is returned in part to the heart by the hypogastric veins. It must be borne in mind, however, that a similar enlargement may be noticed in extreme and protracted ascites from any cause, owing to the pressure of the fluid on the vena cava inferior. In this case, however, there is usually also varix of the veins of the lower extremities.

6. Hæmorrhoids not uncommonly result from the same cause as the enlargement of the abdominal veins, and often precede the other signs of portal obstruction. Hæmorrhoids, indeed, in a large number of cases, are due to obstructed portal circulation, and you will do well to remember that neglect of this fact has too often led to serious, and even fatal, results. The blood from the rectum must pass through the liver, and the occasional discharge of blood from piles acts as a sort of safety-valve for relieving the overfilled radicles of the portal vein. Remove this safety-valve by operating on the piles, and there is a risk of increased mischief in the liver, ascites, or hæmatemesis, ensuing.[1]

7. Hæmorrhages from the mucous membrane of the stomach and bowels occasionally take place and are sometimes profuse and fatal, even before the occurrence of ascites. In some cases they are followed for a time by great relief. The minute vessels of the mucous membrane rupture, owing to their extreme distension with blood and to the collateral circulation not being sufficiently developed. Epistaxis, purpuric spots and blotches on the skin, bleeding from the gums, ecchymoses about the punctures of the lancets in cupping, and other hæmorrhages which are obviously independent of a mechanical cause and are probably the result of some altered condition of the blood, are not uncommon in the advanced stages of cirrhosis, and are always of bad omen.

8. Catarrh of the stomach and bowels also frequently occurs

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[1] 'When baffled by untoward circumstances the bowels plague me too, and discharges of blood relieve the headache, and are as safety-valves to the system. I was nearly persuaded to allow Mr. Syme to operate on me when last in England, but an old friend told me that his own father had been operated on by the famous John Hunter, and died in consequence at the early age of forty. His advice saved me, for this complaint has been my safety-valve.'—*The Last Journals of David Livingstone*, 1874, vol. iii. p. 124.

in the course of cirrhosis, the congested mucous membrane being excited to inflammation by causes which would otherwise be inert. Their occurrence is marked by abdominal tenderness, pain and vomiting after food, and obstinate diarrhœa, with more or less fever. In such cases we often find after death the typical anatomical characters of catarrhal inflammation with hæmorrhagic erosions in the mucous membrane of the stomach, appearances which by the inexperienced are not unfrequently believed to result from some irritant poison. Like hæmorrhage, attacks of diarrhœa in the course of cirrhosis are often salutary and ought not to be hastily checked. More than once I have known ascites induced by the injudicious use of astringents. Where waxy degeneration coexists with cirrhosis, there may be profuse and obstinate diarrhœa from waxy disease of the bowels.

9. Pain in the region of the liver is not a prominent symptom of true cirrhosis. In the early stage there is sometimes a dull heavy pain with some tenderness in the right hypochondrium arising from congestion, and throughout the disease there may be acute pain and tenderness of a temporary nature resulting from intercurrent attacks of peri-hepatitis, but in the intervals of these attacks there is but little pain or tenderness in the region of the liver.

10. Decided jaundice is a rare, and when persistent a bad, symptom in cirrhosis. In the early stage there may be jaundice from congestion; but when jaundice shows itself subsequently it is usually the result of some complication, such as catarrh of the ducts, or enlarged glands in the fissure of the liver compressing the bile-duct. Persistent jaundice may also show itself in the advanced stages of the malady, and be associated with hæmorrhages, dry brown tongue, offensive breath, and restlessness. These symptoms are always ominous, even where there is no ascites; they are often followed by cerebral symptoms. Dr. Fagge, however, has observed a case of cirrhosis with persistent jaundice for seven years, in which the patient died at last of hæmatemesis.[1] (See also Case C. p. 249.)

But although decided jaundice is rarely met with in cirrhosis, there are few patients who do not throughout the disease present a persistent sallowness of the complexion, with a dark areola round the eyes, and yet the motions are coloured by bile and the urine contains little or no bile-pigment. Care must be taken not to confound with this sallowness the bronzed appearance of the face from exposure to the sun in hot climates, or the waxen complexion of anæmia. The combination of this sallowness with sunken features and venous stigmata on the cheeks constitutes the characteristic physiognomy of cirrhosis.

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[1] Guy's Hosp. Rep. 1875, vol. xx.

11. The digestive functions are sometimes in a tolerably normal condition, but more commonly there is loss of appetite, with flatulence and constipation, morning sickness, or the symptoms of gastro-enteritis already referred to.

12. The urine is almost invariably very scanty, high-coloured and acid, and deposits large quantities of pink, dark red, or brownish urates. Even when there is no jaundice, it always contains much abnormal pigment, closely allied in composition to bile-pigment. These characters are so constant that the secretion of pale urine, remaining clear after cooling, would be a strong argument against ascites in any case being due to hepatic disease. Albuminuria is sometimes present, owing to concurrent Bright's disease of the kidneys; but you must remember that a large quantity of fluid in the peritoneum may lead to the appearance of albumen in the urine independently of any disease of the kidneys, the albumen disappearing on the removal of the pressure on the renal veins by paracentesis.

13. Cerebral symptoms, such as drowsiness, delirium, coma, and convulsions, frequently supervene in the advanced stage of cirrhosis. They are often associated with jaundice and hæmorrhages, and they are a contraindication to paracentesis, as they are usually aggravated by a withdrawal of the fluid.

14. In all cases the advance of the disease is marked by progressive emaciation and debility. In consequence of the obstruction of the portal vein, the intestinal absorption of nutritive material is diminished and then suspended, while the blood-forming functions of the liver and spleen are more or less impaired. In many cases the patient dies by exhaustion, the intellectual faculties remaining clear to the last. At other times death is due to pneumonia, œdema of the lungs, or acute peritonitis, or is preceded by jaundice and the symptoms of general blood-poisoning already mentioned.

15. The diagnosis of true cirrhosis will be assisted by remembering the circumstances under which it occurs:—

a. Age. Cirrhosis is chiefly met with in adults between the ages of 35 and 60. It is extremely rare under the age of 25, but it has been met with even in young children; and Case CXI. is an example of the disease in a child aged 9.

b. Sex. It is usually said to be more common in males than in females, but my experience in London leads me to think that there is not much difference in this respect.

c. Habits. In almost all cases there is a previous history of spirit-drinking, and especially a habit of taking spirits or strong wine in an undiluted form and on an empty stomach. It is very rare indeed that true cirrhosis results from any other cause, for although it occurs occasionally in young children[1] (and even, it is said, in some of the lower animals, such as cattle and pigs) it is possible that the disease in many of these cases is one of the other forms of chronic atrophy to which I have called your attention. In young children the disease is sometimes traceable to inherited syphilis, while Case CXI. shows that the apparent exceptions may support the rule, and that even in early life true cirrhosis may result from the abuse of alcohol.

d. Occupation. From what has been stated it is not surprising that the disease is particularly prevalent among publicans and sailors. Still the statement recently put forward on high authority that the disease is rare among the upper classes in society is quite contrary to my experience.

e. Gout. Cirrhosis is very often found in connection with gout. The condition of liver which developes gout renders it liable to suffer from alcohol, even in small quantity.

The clinical characters of the other forms of chronic atrophy are similar to those of true cirrhosis as above described, but I may now mention to you the circumstances which during life may often enable you to distinguish them, although their distinction will not materially influence either prognosis or treatment.

II. The contraction which results from mechanical obstruction of the circulation differs from true cirrhosis in the following respects:—

1. There are the previous history and the existing physical signs of serious disease of the heart or lungs.

2. Dyspnœa precedes the ascites.

3. Although the ascites may preponderate over the signs of dropsy elsewhere, it is preceded by œdema of the legs, which persists. (See Case LV. p. 146.)

4. The existence of true cirrhosis will be rendered still more improbable if there be an absence of any history of spirit-drinking, or of alcoholic dyspepsia.

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[1] See Appendix; also Frerichs, op. cit. vol. ii. p. 60; Dr. Griffiths, Path. Trans. vol. xxvii. 186.

III. The atrophy of the liver in which the organ presents a nodulated outer surface like that of true cirrhosis, but which has no increase of the fibrous tissue, and in consequence has its tissue either of natural consistence or softened instead of being preternaturally dense, cannot be distinguished from true cirrhosis by any clinical characters with which I am acquainted. In Case CXII. which was an example of this form of disease, there was no history of spirit-drinking, so that possibly this negative character may be of some use in diagnosis.

IV. What is called simple induration of the liver, which results from repeated attacks of peri-hepatitis, differs from true cirrhosis in the following particulars.

1. When the edge of the liver can be felt, it is usually smooth and hard, but an exception must be made with regard to those cases which have a syphilitic origin, and where, as already stated, the surface of the liver may be marked by projecting nodules separated by deep fissures.

2. Pain and tenderness in the region of the liver are greater and more constant than in true cirrhosis.

3. The venous stigmata on the cheeks and the morning sickness of alcoholic dyspepsia are often absent.

4. The circumstances under which simple induration is known to occur are important in the diagnosis. Thus there is :—

a. A clear history of constitutional syphilis, or

b. A previous history of local or general peritonitis, of ulceration of the stomach, or of inflammation of the right pleura.

c. An absence of any history of spirit-drinking.

d. Occasionally simple induration is met with in cases of valvular disease of the heart, in conjunction with the second form of chronic atrophy already referred to.

V. Clinically it will always be difficult, and ofttimes impossible, to distinguish 'red atrophy' (see p. 276) from cirrhosis or simple induration. As in these affections, the disease runs a chronic course, there is a great diminution in the area of hepatic dulness, and there are the symptoms of portal obstruction, viz. ascites, enlarged spleen, &c. Severe diarrhœa is common, but there is rarely any jaundice. The surface of the liver, when it can be felt, differs from that of cirrhosis in being smooth, but the chief indications of its existence are the circumstances which precede the symptoms of portal obstruction.

a. There is no history of spirit-drinking.

b. There is no dyspnœa or valvular disease of the heart.

c. There is not necessarily any history of attacks of perihepatitis; but,

d. In many cases there is an antecedent history of ague or malarious remittent fever.

e. In others there has been a history of dysenteric or other ulceration of the intestinal canal.

Treatment of Cirrhosis.

A. If from the presence of the symptoms which I have described to you, there is reason to suspect the existence of *incipient* cirrhosis, the indications for treatment will be similar to those which I have already laid down under the head of congestion of the liver. I must refer you for details to the remarks which I made on that subject, and at present I shall merely mention a few general principles for your guidance.

1. The first and main thing to be done is to put a stop to the patient's drinking habits. Unfortunately it is often very difficult to effect this. The patient may promise obedience to rules, but his cravings for stimulants are irresistible. If alcohol be permitted at all, the allowance ought to be restricted to a pint of hock or claret in the 24 hours, and even these wines are better diluted in soda or seltzer water. But half-measures rarely succeed, and total abstinence is more often successful. The popular prejudice against suddenly cutting off an immoderate quantity of stimulants is, so far as my experience goes, founded on error, unless there be fatty heart, and the patient's craving for stimulants will often be allayed by some bitter infusion in combination with ammonia and ginger.

2. Attention to the diet is also of importance. This ought to consist of such articles as milk, eggs, farinaceous substances, and plainly cooked white fish, poultry, game, and meat. All rich, sweet, and greasy dishes, as well as hot spices and indigestible food of every sort, ought to be strictly interdicted.

3. Regular exercise in the open air ought always to be enjoined.

4. The bowels ought to be kept regularly and freely acting by saline purgatives, such as the sulphates of magnesia, soda, or potash, the bitartrate of potash, Carlsbad or Cheltenham salts, or the mineral waters of Friedrichshall or Püllna. These remedies may be given daily for two or three weeks at a time, and they act best when taken warm on first rising in the morning. Their action is often assisted by occasional doses of calomel, blue pill, or podophyllin, with colocynth or rhubarb.

5. When the liver is enlarged, advantage is often derived from a course of iodide or of bromide of potassium, or of chloride of ammonium (p. 136) or of mercury, while at the same time iodine or iodide of mercury ointment are rubbed into the right hypochondrium. Marked results often follow the internal administration of the green iodide of mercury, in doses of a grain or half a grain three times daily If these remedies fail in reducing theliver, the mineral acids and bitter tonics, and the nitro-muriatic acid bath (see p. 137) may receive a trial.

B. In the *second stage* of the malady no treatment can restore the portion of liver which has been destroyed, or remove the obstruction to the portal circulation. All that can be done is to counteract the effects of the disease, to relieve the engorgement of the radicles of the portal vein, and support the patient's strength by appropriate means, with the hope that the progress of the morbid process may be stayed, and that in time a collateral circulation will be established, by which the portal blood will reach the right side of the heart.[1]

1. You must be still guided by the same rules of treatment as in the early stage with regard to alcohol, diet, purgatives, &c. You must beware of checking spontaneous attacks of diarrhœa, unless it be excessive.

2. Tonics, such as nitro-muriatic acid, gentian, nux vomica, strychnia, or cascarilla, may be given from time to time, to improve the appetite, digestion, and general strength.

3. Ascites will often call for treatment. The remedies most useful for this purpose are :—

a. Purgatives, and those ought to be selected which have most power in increasing the watery exhalation from the mucous membrane of the bowels, such as the salines and mineral waters already referred to, the compound jalap powder, and gamboge. An excellent purgative is an electuary composed of compound jalap powder and confection of senna. These purgatives ought always to be given in the morning before food, so as not to sweep away the food which has been digested but not assimilated. Drastic purgatives, such as elaterium and croton oil, must be given with some caution, for obstinate enteritis is one of the natural results of the disease, and sometimes causes death by exhaustion.

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[1] When the portal vein is obstructed, a collateral circulation may be established by four different channels, viz. :—

1. The blood in the inferior mesenteric veins may be carried by the inferior hæmorrhoidal to the hypogastric veins, and so returned to the heart.

2. By the enlargement of certain branches of the portal vein which pass between the folds of the falciform ligament from the liver to the abdominal parietes, and there anastomose with the epigastric and internal mammary veins.

3. By the enlargement of twigs of the portal vein and of the veins in the capsule of the liver which open into the diaphragmatic and œsophageal veins.

4. By newly formed vessels in the adhesions between the liver and the diaphragm and abdominal parietes. In advanced cirrhosis it is not uncommon to see a large vein emerge abruptly just below the right false ribs, and pass up in a varicose condition over the chest.

b. Diuretics are commonly prescribed in conjunction with purgatives, and different combinations of them may be tried. You may give the acetate or bitartrate of potash, or the iodide of potassium, or the chloride of ammonium, or the benzoate of ammonia, in combination with spirit of nitrous ether, digitalis, and decoction of fresh broom-tops. The perchloride of mercury with digitalis, or the compound digitalis pill (containing blue pill, squill, and digitalis) sometimes answers well. The digitalis pill is one that has enjoyed a long and merited reputation, but it is far more useful in the treatment of cardiac than of hepatic dropsy. Diuresis will also sometimes be induced by fomenting the abdomen and loins with an infusion of digitalis of about four times the Pharmacopœia strength. It is worth noting also that copaiba will occasionally succeed in increasing the flow of urine and removing hepatic dropsy after all other diuretics have failed. The best form is the resin, which may be given in doses of 15 gr. three times a day.[1] By acting on the kidneys there can be no doubt that we sometimes succeed (as in Case CLXII., Lect. XII.) in diminishing or retarding the increase of the ascites; but when the ascites is already great, it must be confessed that diuretics are of little avail, and that in fact they fail to increase the flow of urine.

c. Tonics, such as quinine and iron, sometimes appear to remove hepatic dropsy after all the more common measures have failed. According to Dr. Bristowe,[2] they are the only remedies that have succeeded in his hands; and although this has not been my experience, I have seen good results from a combination of the tincture of perchloride of iron with digitalis, or from the tartrates of iron and potash with digitalis.

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[1] The resin is to be well rubbed with twice its weight of compound powder of almonds, and an ounce of water added, so as to form an emulsion. See Lancet, Feb. 27, 1869; Trans. Clin. Soc. 1869, vol. iii. p. 26; Guy's Hosp. Rep. 1876, vol. xxi.

[2] Trans. Clin. Soc. vol. ii. p. 12.

d. Collections of fluid in the abdomen are sometimes rapidly absorbed after the abdomen has been kept covered for some time with lint smeared with linimentum hydrargyri, to which belladonna may be advantageously added if there be much pain. This good result, however, is more likely to ensue when the fluid results from chronic inflammation than when it is caused by portal obstruction.

e. Notwithstanding the use of these various remedies, the ascites too often slowly increases, and sooner or later the belly attains such a size as to seriously embarrass the breathing and necessitate a recourse to paracentesis. The tapping may have to be frequently repeated, and the rule commonly laid down is always to delay it as long as possible, until in fact there is danger of the respiratory function becoming seriously interfered with by the pressure of the fluid. It is argued that, as the fluid collects again rapidly, the frequent repetition of the operation must increase the patient's exhaustion, owing to the great drain of albumen from the blood which the reaccumulation entails. But there are good grounds for reconsidering this rule. The operation when delayed until the last is often followed by rapid sinking with typhoid symptoms. On the other hand the advantages of early tapping are, that by removal of pressure the establishment of a collateral circulation through the more healthy portions of the liver itself, as well as through the veins of the abdominal parietes, is promoted. Secondly, the functions of important parts which had been impaired or arrested by the pressure are restored. Not only are the lungs relieved, but by the removal of pressure from the portal and renal veins, assimilation and the secretion of urine are increased. I have known hæmorrhage from the bowels arrested by paracentesis in cirrhosis, and it is a common observation that patients with much ascites, who, notwithstanding the most powerful diuretics, have been passing only a small quantity of urine containing much albumen, will, after paracentesis and independently of drugs, void large quantities of urine free from albumen. And thirdly, diuretic and other remedies, which, when the abdomen is full of fluid have produced no effect, probably from not being absorbed, will often after paracentesis act powerfully, and thus retard or prevent the reaccumulation of fluid in the peritoneum. As soon, therefore, as the abdomen becomes moderately distended with fluid, and the remedies which I have mentioned to you fail to produce any effect, I would recommend you to lose no time in having recourse to paracentesis.[1] Even should the fluid reaccumulate repeatedly you need not despair. In Case CXV. the patient was tapped four times, and after the fourth tapping there was no accumulation of fluid; while not long ago a case of cirrhosis was reported by Dr. Lyons of Dublin in which the patient was tapped thirty-six times at intervals of three weeks or a month, from 14 to 16 quarts of fluid being drawn off on each occasion; one year after the last tapping, the ascites was stationary.[2] In performing the operation, there are one or two particulars which I would advise your attending to. First, the trocar ought to be of much smaller size than that which is commonly employed, and flattened instead of rounded; the wound resulting from such an instrument is closed without difficulty. And secondly, it is better not to attempt to empty the peritoneum at each operation; mischief sometimes results from the pressure necessary to effect this, and all the good effects of the operation will be obtained, although two or three pints of fluid remain. In connection with this subject I would call your attention to some interesting observations of Professor Leudet of Rouen,[3] who advocates the employment of an exploratory trocar for drawing off the fluid. In two cases which he has published the instrument was inserted into a protrusion of the umbilical cicatrix. This procedure had these advantages: the integuments at the seat of puncture were very thin; the opening readily closed; and the instrument did not enter the general cavity of the peritoneum.

f. When there is much œdema of the legs, as well as ascites, both may be relieved by acupuncture of the legs, or by making an incision through the skin into the areolar tissue about an inch above the inner ankle of each leg, followed by poultices, according to the plan recommended many years ago by a distinguished physician of this (St. Thomas's) hospital, Dr. Mead. The quantity of serum which will sometimes drain away from these punctures or incisions, to the great relief of the patient, is surprising.

4. Intercurrent attacks of peri-hepatitis may require local depletion, cataplasms, and opium.

5. Attacks of gastritis will demand sinapisms and blisters

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[1] For additional remarks on the advantages of early tapping in ascites, see paper by Dr. John McCrea, Dub. Journ. of Med. Sc., Aug. 1873; also Brit. Med. Journ. 1873, vol. i. pp. 185, 250.

[2] Brit. Med. Journ. 1873, i. 185.

[3] Clin. Méd. Paris, 1874, p. 557.

to the epigastrium, with ice and lime water, or bismuth and hydrocyanic acid internally. The diet should consist of milk and farinaceous articles; and when the vomiting is urgent, it should be restricted to milk. Wine and spirits, which are often taken under such circumstances, and which may give temporary relief, always do harm. If their employment is considered absolutely necessary, they ought to be given by the rectum. After all other measures have failed, four or five grains of calomel will sometimes at once arrest the sickness.

6. In enteritis it may be necessary to apply a few leeches round the anus, and to administer the vegetable or mineral astringents with opium, and in particular the acetate of lead with morphia, but it is inexpedient to check the purging too hastily or too completely.

7. When copious hæmorrhage occurs from the stomach or bowels, the remedies indicated are ice, ergot, a combination of saline purgatives and astringents, such as a mixture containing sulphate of magnesia, tannin, and sulphuric acid, and the application of leeches round the anus.

8. Flatulence is often a source of so great distress, and aggravates so much the dyspnœa arising from the ascites, as to call for treatment. It will often be relieved by the various ethers and the essential oils of peppermint, anise, or cajeput, by vegetable charcoal, or by galbanum and assafœtida. Inasmuch, however, as it is probably due to decomposition from deficient or deteriorated bile, those remedies will be found most useful which act by checking decomposition, such as creasote, turpentine, or carbolic acid (see p. 215).

9. When cerebral symptoms and other signs of blood-poisoning supervene, no treatment will probably be of any avail, but I have known great, though temporary, improvement result from calomel and saline purgatives, and from blisters to the scalp.

The principles of treatment which have been recommended for cirrhosis are applicable to the other forms of chronic atrophy of the liver, with the following modifications.

a. In atrophy of the liver resulting from disease of the heart, the treatment of the symptoms of obstructed portal circulation must be subsidiary to that of the more important primary disease in the chest. Diuretics are more effectual in removing the dropsy, and especially the compound digitalis pill (see p. 287); but even here, although alcoholic stimulants will be more necessary than in true cirrhosis, they must be given with caution (see p. 135).

b. In cases with a marked syphilitic history, and where there is reason to infer the presence of syphilitic peri-hepatitis, mercury and iodide of potassium may be expected to be of service.

I shall now narrate to you a few cases of chronic atrophy of the liver, in illustration of the remarks which I have made respecting its pathology and treatment. The first is a good example of true cirrhosis from spirit-drinking.

CASE CIV.—*History of Spirit-drinking and Symptoms of Portal Obstruction—Dense fibrous granular Liver—True Cirrhosis.*

Thomas B——, aged 52, a butcher, admitted into Middlesex Hosp. April 30, 1867. His father and mother had lived to an advanced age; a brother and sister had died of consumption. He was a large, stout man, had always enjoyed good health until about two years ago, when he began to suffer from flatulence, and during last year he had also complained of shortness of breath, disturbed sleep, chilliness, and occasional palpitation; at same time he had noticed some swelling of legs and abdomen. He thought that this swelling had gone away after two or three weeks, but about four months ago it had reappeared, and had since increased considerably. He said that it had first reappeared in left leg, but possibly it was not greater in this situation than elsewhere, and his attention had been more directed to it owing to a vesication which appeared over left ankle. He had never had hæmorrhoids, but on several occasions during last two years he had vomited about a teacupful of black blood. His habits had been always intemperate; he had drunk freely both spirits and beer.

On admission patient exhibited an emaciated, sallow countenance, with a slightly jaundiced tint of conjunctivæ and venous stigmata on cheeks. Great œdema of lower extremities and scrotum and evidence of a large accumulation of fluid in peritoneum, umbilicus being quite obliterated, and girth of abdomen being 46 in. There was also considerable enlargement of subcutaneous veins of abdomen, especially on right side. Hepatic dulness diminished in right mammary line, being less than 3 in. (see fig. 27, p. 278); the splenic dulness was increased, measuring vertically 4 in. No jaundice, except the slight icteroid tint of conjunctivæ above referred to. No tenderness of abdomen, nor vomiting; tongue moist, with a white fur; bowels very costive, often not acting for a week; heart's action feeble, but dulness and sounds normal. Pulse 120. Crepitus, at some places rather fine, audible over lower half of both lungs, back and front, but no decided dulness, nor tubular breathing; respirations 36. Urine acid, no albumen, but a decided reaction of bile-pigment: specific gravity 1032.

Patient was treated with purgatives and diuretics, but no improvement took place in his condition; on contrary dropsy increased in legs, and on May 17 girth at umbilicus was 47½ in.; but head, neck, arms, and chest were free from œdema. Dyspnœa increased, and mucus much streaked with red blood was expectorated. Urine was very scanty, but free from albumen, having been tested daily till May 17. Patient gradually grew weaker, and on evening of May 18 he became suddenly much worse; pulse very quick (136) and irregular: very restless and delirious, and tongue dry and brown; bowels relaxed. There was no increase of dyspnœa, no lividity of face, and no alteration in physical signs of lungs. Patient continued much in same state until his death at 11 A.M. of following day.

On post-mortem examination, great lividity and puffy swelling of face and neck, and brownish staining of integuments along course of subcutaneous veins. Heart was healthy; no appearance of pleurisy or pneumonia, but both lungs very congested and œdematous; peritoneum contained several gallons of slightly turbid, straw-coloured serum. Notwithstanding absence of albuminuria, both kidneys much enlarged, right weighing 9 oz. and left 9¼ oz.; capsules non-adherent, and surfaces smooth; cortex hypertrophied, flabby and soft, and mottled with numerous small, dark, ecchymotic spots; renal epithelium loaded with fine granules, but contained little oil. Spleen large and soft, and weighed 7 oz.

Liver very small and quite hidden below right ribs. It measured 9 in. from right to left; antero-posterior diameter of right lobe was 6½ in. and of left 5 in.; weight was 43¾ oz. avoird. Outer surface nodulated and granular, and presented the typical characters of cirrhosis. Capsule not thickened nor adherent; structure much increased in density, and on section presented islands of light yellow secreting tissue, cells of which contained much oil, surrounded by broad bands of firm white tissue including bile-ducts and hepatic vessels. Gall-bladder distended with 4 oz. of thin, watery, greenish bile, in which were a large number of minute black concretions of inspissated bile, several of which were also found in cystic, hepatic, and common ducts. Fæces in intestines were coloured yellow.

Case CV. is another illustration of true cirrhosis arising from spirit-drinking. The cerebral symptoms were due to the complication of disease of the kidneys. The attacks of jaundice from which the patient suffered were probably the result of catarrh of the bile-ducts; the fact of their being preceded on each occasion for some days by vomiting pointed to antecedent irritation in the stomach and duodenum. That the jaundice was independent of the cirrhosis is shown by its almost disappearing before death, although the symptoms of portal obstruction increased.

CASE CV.—*History of Spirit-drinking—Cirrhosis of Liver—Nephritis—Epileptiform Convulsions, and Death by Uræmia.*

Derby H——, aged 45, admitted into Middlesex Hospital on Ocober 15, 1867. He was a barman, and for six or seven years he had been in the habit of drinking large quantities of gin, and during last six months he had been very often intoxicated. About two years before admission he began to suffer from attacks of vomiting, followed after a few days by temporary jaundice, but not accompanied by pain like that of biliary colic. Twelve months before admission vomiting became more constant and urgent, especially in the morning, and was accompanied by diarrhœa and by a persistent pain in region of liver and slight jaundice; vomited matters often contained blood. He had also several attacks of epistaxis, one of which was so severe that he was taken to Charing Cross Hospital, where his nostrils were plugged. Six weeks before admission he had a severe fit of epileptiform convulsions, in which he bit tongue deeply. In following three weeks he had four similar attacks, last of which was followed by jaundice, and a condition resembling delirium tremens, which continued up to time of admission.

On admission, patient's mind very confused, expression stupid, and considerable stupor. Conjunctivæ and whole surface of body rather deeply jaundiced, and very slight pitting of lower extremities. Tongue dry and brown down centre, and on left margin, where it had been bitten in one of fits, there was a deep ulcer. Motions contained bile. There was now neither vomiting nor diarrhœa, but there was evident tenderness on pressure below right ribs. Liver could not be felt, and hepatic dulness in right mammary line was diminished, not exceeding 3 in. At margin of ribs, however, corresponding to situation of gall-bladder, a distinct rounded tumour could be felt, about size of a hen's egg. Abdomen much distended from tympanites, measuring at umbilicus $32\frac{3}{4}$ in.; no sign of ascites could be discerned, but splenic dulness increased, and subcutaneous veins of abdomen enlarged. Pulse 84, feeble but regular; cardiac dulness slightly increased towards left, measuring transversely $2\frac{1}{2}$ in.; sounds weak, but no bellows-murmur. No dyspnœa nor cough, and physical signs of lungs normal. Urine very dark, like porter; sp. gr. 1020; it contained abundance of bile-pigment and a trace of albumen; under microscope a few blood-corpuscles were seen, but no casts. Temperature 101·2°.

Patient was treated with laxatives, diaphoretics, and diuretics, and a simple diet without any stimulants; he had also several warm baths, and mustard poultices applied to back of neck and over region of liver. Under this treatment, after two or three days, he began to improve, and at end of ten days he was able to get up and go about ward. Jaundice and tympanites were greatly diminished; tumour in region of gall-bladder had disappeared, tongue was moist and clean, appetite had returned, and temperature was normal. Urine, however, still contained a slight trace of albumen ($\frac{1}{20}$), and patient's memory was confused as to dates. With exception of a painful inflammatory swelling in meatus of left ear, lumbar pain, an attack of vomiting with slight epistaxis on Nov. 22, and a return of stupor with dryness of tongue during last week of November, this improvement lasted until Dec. 12. About this time he became much weaker, and very restless and delirious at night; abdomen had again enlarged, measuring at umbilicus $35\frac{1}{4}$ in., and there was now unmistakable evidence of a small quantity of fluid in peritoneum. There was also considerable œdema of lower extremities, and a distinct systolic blowing murmur at base of heart. The albumen in urine, however, was not increased; temperature was only 96° F., and there was scarcely any jaundice. Treatment consisted in administration of liquor ammoniæ acetatis with acetate of potash, tincture of digitalis, and decoction of broom-tops with mild laxatives, and subsequently compound jalap powder and croton oil, and application of mustard and linseed poultices to loins. Ascites, however, gradually increased until abdomen measured 40 in., but after patient kept his bed dropsy of legs almost disappeared, and there was no trace of dropsy of face or upper part of body. Enlargement of abdominal veins increased, urine was scanty and smoky, and contained a much larger amount of albumen ($\frac{1}{6}$), with epithelial and blood-casts. Tongue dry and brown; motions and urine passed in bed; temperature throughout was rather below normal standard. For three or four days before death, which took place on Dec. 23, much noisy incoherent delirium with spasmodic twitchings of extremities, but no general convulsions.

The appearances found after death were as follows:—Slight jaundice of integuments. Several quarts of clear straw-coloured serum in peritoneum. Liver very small, measuring only 9 in. from right to left, and 6 in. from back to front, in right lobe; greatest thickness $3\frac{1}{4}$ in.; weight 44 oz.; outer surface coarsely granular; glandular substance extremely dense, and consisting of firm bands of fibrous tissue, including bile-ducts and obliterated vessels, and enclosing islets of yellow secreting tissue, cells in which were loaded with yellow pigment and oil. Spleen large, 7 oz., firm. Pancreas very large; and indurated from presence of an unusual amount of fibrous tissue. Extreme catarrhal inflammation, with hæmorrhagic erosions, of mucous membrane of stomach. Both kidneys enlarged, weighing together 14 oz.; capsules separating readily; surfaces smooth; cortices hypertrophied; marked injection of straight vessels in pyramids and of Malpighian bodies; renal tubes gorged with granular epithelium. Considerable hypertrophy of left ventricle of heart, and a vegetation the size of a hemp-seed on ventricular surface of each of aortic valves. Much hypostatic congestion of both lungs, and a few old adhesions over surface of left. Much serous fluid containing urea beneath arachnoid and in lateral ventricles of brain.

In the following case there was every reason to believe from the history that the atrophy of the liver was due to true cirrhosis, but the only indications of portal obstruction were copious hæmatemesis, hæmorrhoids, and slight enlargement of the spleen. In several instances, however, I have known persons die suddenly of hæmatemesis, who had previously been sufficiently well to perform their duties in life, and in whose bodies well-marked cirrhosis of the liver has been found after death. The treatment, though successful, was not such as I would recommend to you in similar cases (p. 290).

CASE CVI.—*History of Spirit-drinking—Contracted Liver—Copious Hæmatemesis—Delirium Tremens.*

Eliza D——, aged 29, a person in good circumstances, admitted into Middlesex Hosp. Feb. 5, 1867. Had been married for five years, and was mother of two children. Ever since her marriage, and perhaps before, she had been addicted to spirit-drinking. This had led to a separation from her husband. For a long time she had been in the habit of getting drunk two or three times a week. She had not suffered, however, from pain and vomiting after food.

Early in morning of day of admission, after a restless night, patient vomited mucus streaked with blood, and an hour afterwards she brought up a large quantity of pure blood. The medical man who was called to see her said that there were at least two pints. Brandy and ice were administered, but she vomited more blood, and on trying to get up, she fell down insensible, and was brought to hospital at noon.

On admission, skin was noted to be sallow, although there was no decided jaundice. Tenderness at epigastrium; hepatic dulness diminished, not amounting to 3 in. in right mammary line; splenic dulness increased; several small hæmorrhoids about anus; but neither ascites nor enlargement of abdominal veins. Urine contained a trace of albumen; a faint systolic bellows-murmur over heart, but cardiac dulness and impulse not increased; physical signs of lungs normal; no œdema of legs.

Patient was treated with gallic acid and opium, and with ice and milk, but no stimulants. For several days vomiting continued, but, except on day of admission, vomited matters contained no blood. Bowels did not act for five days after admission; enemata then brought away a large quantity of tar-like matter. For several days after admission patient suffered from a severe attack of delirium tremens, but by Feb. 11 this had quite subsided, and she was able to eat and retain solid food. Vomiting did not recur, and on Feb. 16 she was discharged.

The interest of Case CVII. consisted in this, that the patient appeared to be in good health until the sudden occurrence of copious hæmatemesis, which was speedily followed by ascites and death.

CASE CVII.—*Cirrhosis—Persistent Hæmatemesis the first notable symptom—Ascites.*

Thomas B——, aged 53, a bargeman, adm. into St. Thomas's Hosp. April 18, 1874. Father alive and in good health, nearly 70; mother died at 43. He was an only child, and up to age of 43 he had been repeatedly in tropics and had suffered from dysentery and malarious fevers, and pain in region of liver. He had not led a temperate life, but, with exception of constipation and some distension of abdomen which came and went, he had enjoyed good health until early in morning of March 24, when he vomited several pints of dark blood. Next day he again vomited a large quantity of blood, and from then until admission he had continued to vomit food and mucus night and morning. Abdomen had enlarged steadily, and bowels been confined, but no pain. Legs began to swell a few days before admission.

Admitted on account of a return of hæmorrhage. Countenance sallow with enlargement of cutaneous veins of cheeks, but conjunctivæ white. Slight œdema of legs. Abdomen greatly distended from fluid in peritoneum, measuring at umbilicus 40½ in.; not tender. Abdominal veins much enlarged. Hepatic dulness commenced half-an-inch below right nipple; lower margin could not be made out. Constant vomiting; food at once rejected; vomited matter also contained much blood, partly in coagula and partly mixed with viscid mucus; motions passed after medicine black and very offensive. Tongue pale, devoid of epithelium, fissured and dry down centre; much belching of gas. Urine scanty, 1035, high-coloured and loaded with lithates. Pulse 104, regular but feeble; heart displaced upwards; no abnormal murmur. Frequent cough, and a few bronchial râles over lungs. Sleep much disturbed. Temp. 97·8°.

Was ordered ten grains of calomel followed by citrate of magnesia, and a mixture of bismuth and soda, with milk and soda-water. Subsequently small quantities of stimulants were given, and the subcutaneous injection of ergotin was tried to check the hæmorrhage. Hæmorrhage, however, from stomach and bowels persisted, and patient became daily weaker and more anæmic. On morning of April 24 he was extremely low, temperature being only 96·3°, and at 11 P.M. he died.

Autopsy.—About 20 pints of serous fluid in peritoneum. Peritoneum generally thickened, and numerous firm adhesions connecting liver with diaphragm, stomach, and other parts. Spleen large, and also adherent. Capsules of spleen and liver, and omentum and mesentery, much thickened. Liver very small; after removal of capsule, surface nodulated; on section firm fibrous bands separated yellow islets of secreting tissue. Black tarry matter in colon. Heart and kidneys healthy. Lungs adherent at bases and œdematous. Brain anæmic.

Many of you watched the patient, whose case I am now about to relate, with much interest, and it is to be regretted that no opportunity was afforded for examining the condition of his liver after death, inasmuch as there was considerable obscurity as to the cause of the atrophy. The signs of obstructed portal circulation, however, were well marked, and the circumstance of hæmatemesis preceding the other signs of obstruction, for several years, is interesting in connection with what was observed in Cases CVI. and CVII.

CASE CVIII.—*Chronic Atrophy of Liver—Ascites—Hæmatemesis and Bloody Stools.*

James T——, aged 38, was adm. into Middlesex Hosp. Aug. 20, 1866. For six years he had been a brewer's drayman, and been accustomed to drink a good deal of ale, but not spirits; before that he had been a farm labourer and had drunk little of alcohol in any shape. He had never suffered from ague or rheumatic fever, but at age of 18 he had been laid up for a year with a cough and debility, and had been told at the Reading Infirmary that he had consumption. He recovered, however, and remained well until eight years before admission, when he received a kick from a horse on right side. He did not take much notice of this at the time, and followed his work for five or six weeks afterwards. Whether owing to this injury or not, he then began to suffer from great pain and tightness at epigastrium, with constipation. He took some aperient medicine, which operated, but on following day he vomited a large quantity of clotted blood, and for a week afterwards he continued to pass blood per anum. This left him very weak, but relieved pain, and he returned to work. After this he had a similar attack about once a year, only difference being that quantity of blood lost was less than it had been the first time. On each occasion vomiting of blood had been preceded for several days by great headache, nausea, and pain in abdomen. Last attack had occurred four months before admission. In spring of 1865 he had been for several weeks in a metropolitan hospital for hæmorrhage. Shortly after leaving that hospital, in April 1865, abdomen became swollen, and subsequently, his legs. He drank 'broom-tea,' and swelling subsided, but a month before admission it increased again.

On admission, patient was emaciated and sallow, but conjunctivæ were white. Abdomen greatly distended from fluid in peritoneum, and veins of abdominal wall unusually large and distinct, but nowhere tenderness, except on pressure over right hypochondrium. Liver could not be felt, and hepatic dulness in right mammary line measured only 2½ in., an observation which was subsequently confirmed after paracentesis. Tongue slightly furred; bowels costive; slight flatulence after meals; appetite good. An anæmic bellows-murmur over sternum; cardiac dulness not increased. Respirations 20, and easy; at bases of both lungs a little fine crepitation. Upwards of 40 oz. of urine were passed daily; it was dark, but contained neither albumen nor bile-pigment. Moderate œdema of both legs.

Treatment consisted in purgatives and diuretics, and for some time bromide of potassium in five-grain doses three times a day. A generous diet was allowed, but no stimulants. At first there was considerable improvement, and girth of abdomen was reduced 2 in.; but about middle of September swelling increased again, and on 28th abdomen measured 42 in., integuments over it were tight and glistening, and urine was reduced to about a pint daily; respirations 32, and considerably embarrassed. On Sept. 30 patient suffered much from pains in abdomen, and during following night he began to pass frequent motions containing much black blood. On Oct. 6 patient had still diarrhœa with bloody stools; abdomen had increased to 44 in.; legs also were swollen, and great orthopnœa. Paracentesis was performed, and 17 pints of fluid were drawn off; fluid clear, straw-coloured, and alkaline; specific gravity 1012; it contained a large quantity of both chlorides and albumen. The operation gave great relief to breathing; anasarca of legs diminished; urine rose to two pints; and blood disappeared from motions. Two days, however, had not elapsed before swelling was again noticed to be increasing, and on Oct. 15 abdomen measured 42 in., œdema of lungs had extended, and patient suffered much from dyspnœa and cough. On Oct. 21 vomiting came on; vomited matters contained a good deal of blood; motions again also contained blood. These symptoms continued until Oct. 24, when patient insisted on leaving hospital. He was removed to Egham, where he died the same evening. His friends would not permit his body to be examined.

In Case CIX., although the liver was really much atrophied, it appeared during life to be enlarged from being displaced downwards by an accumulation of serous fluid between its upper surface and the diaphragm.

CASE CIX.—*Small Cirrhotic Liver displaced downwards and simulating Cancer—Ascites and Jaundice.*

Agnes F——, 32, single, needlewoman, adm. into Middlesex Hosp. May 29, 1869. About a year before admission began to suffer from loss of appetite, weakness and languor, pains in abdomen, and fulness after eating. After four months she had, in addition to these symptoms, nausea, eructations of sour frothy fluid, and retching in morning. Four weeks before admission a doctor, who had been called to see her for a wound of forehead, discovered that she had 'dropsy in the stomach.' From the statements of her friends there seemed little doubt that she had been frequently intoxicated.

On admission was thin and weak, and had considerable œdema of lower extremities. Peritoneal cavity distended with fluid, girth of abdomen at umbilicus being 36½ in. Superficial veins of abdomen enlarged. On abrupt palpation, lower edge of what appeared to be an indurated liver could be felt distinctly 3 in. below ribs in right nipple line; contact of finger with this always caused pain. There was also dulness on percussion in right nipple line for 4 in. above lower margin of ribs. Decided jaundice of skin and conjunctivæ. Tongue dry down centre; appetite bad; much thirst; no retching; bowels open, and motions contained plenty of bile. P. 84; resp. 18; heart and lungs appeared to be sound; temperature normal; no rigors nor perspirations. Sleeps badly; expression vacant; and wanders at times. Urine 1023, contained much lithates and bile-pigment, and a minute quantity of albumen. On left temple was a wound with dirty swollen edges, and left cheek was ecchymosed.

After admission, patient continued to get worse. Jaundice diminished, but ascites increased until July 14, when girth at umbilicus was 45½ in. During last week of June she began to vomit food, and to complain of pain in abdomen so severe as to necessitate frequent subcutaneous injections of morphia. Delirium and restlessness continued, and pulse rose to 120, but temperature was always normal. Became gradually weaker, and died on July 22.

Autopsy.—Peritoneum contained many quarts of clear yellow serum, a quantity of which had collected between diaphragm and upper surface of liver. That this arrangement had existed during life seemed probable from there being no other explanation of fact, that liver had been repeatedly felt 3 in. below ribs, and yet was preternaturally small, its weight being only 36½ oz., its extreme length 9 in., and its greatest breadth 7 in. It was a typical example of true cirrhosis. Spleen slightly enlarged, soft and congested. Mucous membrane of stomach intensely congested. Lungs, heart, and kidneys normal.

After the operation of paracentesis, fluid often continues to drain away from the opening for many days, and the patient dies from exhaustion, or from the supervention of peritonitis, and in Case CX. the attempt to close the opening seemed to induce peritonitis. These dangers will be avoided by employing a small flattened trocar, instead of the large rounded instrument in common use (see p. 289).

Case CX.—*History of Spirit-drinking—Cirrhosis of Liver—Ascites—Paracentesis—Patency of opening—Attempts to close it followed by Peritonitis.*

John L——, house-agent, aged 47, adm. into Middlesex Hosp. Dec. 30, 1868. From age of 16 until three years ago had been in habit of drinking on an average upwards of half a pint of spirits daily, besides beer. Left off drinking spirits because it made him vomit his food. After this had better health until Sept. 9, when he was kicked by a horse in private parts, in consequence of which he was laid up in hospital for 2½ months with abscess of scrotum followed by erysipelas of legs. On recovering, he began to complain of pain in both hypochondria, and three weeks before admission abdomen began to swell: more recently legs had become swollen.

On admission, countenance spare and sallow, with stellate veins on cheeks, but no jaundice of conjunctivæ. Considerable œdema of legs, dependent parts of trunk, penis, and scrotum. Much fluid in peritoneum; girth of abdomen, 3 in. above umbilicus, 46 in. Complains greatly of feeling of tightness in abdomen. Abdominal veins enlarged. Hepatic dulness in r. m. l. 3¼ in.; no appreciable enlargement of spleen. Tongue too red; fair appetite; no vomiting; bowels regular. P. 104, weak; heart signs normal. Sibilant râles and prolonged expiration over both lungs. Urine contained a trace of albumen and also of bile-pigment, and mainly passed involuntarily. Two bed-sores over sacrum.

Patient was in such distress from distension of abdomen that on Jan. 1 paracentesis was performed, and 16 pints of clear straw-coloured serum drawn off. Great relief followed operation; but fluid continued to drain away in such large quantity from opening, that, on Jan. 4, at suggestion of my colleague Mr. Moore, this was closed by a needle and twisted suture. Next morning patient complained of nausea and of pain in abdomen, which was also tender. In course of day he vomited frequently, and had all symptoms of acute peritonitis, which continued until death on evening of Jan. 6.

Autopsy.—Fully 12 pints of turbid flaky fluid in peritoneum. Much vascularity and adherent recent lymph in neighbourhood of puncture. Liver small and entirely concealed beneath ribs; but very dense, and weighed 68 oz.; a typical example of true cirrhosis. Spleen adherent and capsule thickened. Both lungs emphysematous, congested posteriorly, and bronchial tubes full of muco-pus. Right side of heart somewhat dilated. Kidneys congested, but otherwise healthy.

Case CXI. was interesting, as being an example of true cirrhosis in a boy aged 9, and also from the circumstance that notwithstanding his youth, the patient had been addicted to stimulants. The diagnosis was embarrassed by the fact that the boy's habits were not ascertained until after his death, by the occasional rise of temperature at night, by the tubercular family history, and by slight crepitation having been noted at the apex of the left lung when the boy was first admitted into hospital. The occurrence of cirrhosis in children has been frequently appealed to, to show that the disease is not due to the abuse of alcohol, but here, as well as in another case recently under my care, what appeared at first to be an exception, was found to confirm the rule. It is worth mentioning also that Wunderlich observed typical examples of cirrhosis in two sisters aged 11 and 12, both of whom, on careful enquiry, were found to have been dram-drinkers.[1] Cheadle, also, has recently recorded a case of extreme cirrhosis in a boy aged 18, who had been in the habit of drinking large quantities of gin.[2] Lastly, Dr. Wilks had under his care in Guy's Hospital, not long ago, a little girl eight years old, suffering from what proved to be a very small hobnailed liver; she had been addicted to drink, having taken as much as half a pint of gin daily.[3] It is possible that the greater activity of the liver in early life may render it more liable to suffer from alcohol than in adults.

CASE CXI.—*True Cirrhosis in a Boy aged 9—Ascites—Paracentesis.*

Henry N——, aged 9, schoolboy, adm. into St. Thomas's Hosp. Sept. 5, 1875. Father alive and in good health; mother died of phthisis, and several brothers and sisters had died in childhood. As far as boy was aware, he had always had good health until about two months before admission. During whole of July he had suffered from sickness and retching every morning. On Aug. 1 he had been sent to sea-side, but the sickness persisted, and abdomen began to swell. After 16 days he returned home; the sickness now ceased, but the swelling increased. From first he had been losing flesh, and bowels had been rather confined.

After boy's death it was ascertained that his father kept a small public-house, and that boy had been in habit of drinking a good deal of wine and water, especially between meals. While in hospital also, he took stimulants with a readiness quite unusual in children.

On admission, boy was emaciated, but abdomen very large, owing to fluid in peritoneum. Girth at umbilicus 32½ in. Abdomen not tender. Lower margin of liver could not be felt; upper margin not too high. Spleen much enlarged; lower end fully 4 in. beyond ribs. No jaundice; no œdema of legs; no albuminuria; no sign of constitutional syphilis, and sounds of heart normal. Temp. occasionally as high as 100·6°. Eats and sleeps well. Tongue normal.

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[1] Niemeyer's Text Book of Pract. Med., Amer. Transl., i. 641.

[2] Brit. Med. Journ. 1871, ii. 545.

[3] Dr. Hilton Fagge, Guy's Hosp. Rep., 1875, Ser. iii. vol. xx.

Boy was at first treated with syrup of iodide of iron internally, while tincture of iodine was painted over abdomen. On Sept. 8, and 9, temp. in evening rose to 102·8° and 103·4° but usually it was under 100°. On Sept. 18 girth at umbilicus had increased to 34¼ in. Citrate of ammonia was now substituted for the iron, and a mercurial plaster was applied to abdomen, while bowels were kept open. Under this treatment ascites rapidly diminished, and on Oct. 5 no trace of it remained; girth at umbilicus was only 24 in., and except that spleen remained large, and he was weak, boy seemed well. He was now treated with iodide of potassium and iron, digitalis, and cod-liver oil. Once or twice he was sick in morning, and on Oct. 24 abdomen seemed to be swelling again, girth being 25½ in. After this swelling rapidly increased until, on Nov. 10, girth was again 34½ in. and tongue red and dry. Bile in urine. Digitalis and iodide of potassium, with aperients, produced no effect; and on Nov. 12 girth 37½ in., integuments of abdomen smooth and shining. Pulse 120; respirations 60, embarrassed. Fifteen pints of clear straw-coloured serum were drawn off by paracentesis, and patient was ordered blue pill, squill, and digitalis, and saline diuretics. The fluid rapidly reaccumulated, and on Nov. 24 girth 38 in.; pain in abdomen and occasional vomiting; considerable œdema of legs; extreme dyspnœa and prostration. Paracentesis again performed, and 16 pints of fluid drawn off, with temporary relief; but next day patient complained of intense pain in abdomen and nausea, and abdomen was refilling; at 7 P.M. collapse came on, and at 11.20 P.M. he died.

Autopsy.—Peritoneum contained 6½ pints of opaque ascitic fluid, containing a few flakes of lymph. Peritoneum generally much injected. Great omentum matted into a mass adherent to adjacent intestines; mesentery thick and œdematous. No tubercle. Liver small; it weighed 21½ oz., normal weight for patient's age being about 32 oz.; outer surface presented typical hobnailed character of cirrhosis; numerous small, irregular, yellowish prominences, separated by pinkish grey depressions. Substance very hard, tough and leathery. On section, yellowish islets of secreting tissue surrounded by fibrous bands. Gall-bladder contained normal bile. Spleen large; weighed 10 oz.; deep red, firm. Mucous membrane of stomach thickened, deeply injected, and with much adherent viscid mucus. Lower part of ileum congested. Peyer's patches and solitary glands of large intestine slightly enlarged. Kidneys large and congested, but structure normal. Heart and lungs normal, with exception of some hypostatic congestion of both lungs and small ecchymoses in subpleural tissue.

The two livers which I now show you appear to me to account for certain differences of opinion still entertained respecting the pathology of cirrhosis. One was taken from the case I have already detailed to you as a good example of true cirrhosis (Case CIV.), the other from the body of the patient whose case I am about to mention to you (Case CXII.).

On the one hand, it is stated that in cirrhosis there is an increase of fibrous tissue, the result of a chronic inflammatory process, and that the secreting tissue becomes atrophied from the pressure exerted on it by this fibrous tissue, or from the conversion of the gland-cells into fibre-cells; while on the other, it is contended that the secreting tissue is simply atrophied, and that the fibrous tissue is not absolutely, though relatively, increased. The former view is the one advocated by Dr. Budd in his work on Diseases of the Liver,[1] and the latter has been put forward by Dr. Beale,[2] and has been adopted by Sir Thomas Watson in the last (fifth) edition of his classical Lectures. A third class of pathologists, among whom may be mentioned Förster, believe that there are two forms of granular cirrhosis, one in which the fibrous tissue is increased, and another where it is not, and this is the view which I have already placed before you (see pp. 275, 284). In one of these two livers the structure is extremely dense, and the fibrous tissue appears greatly increased, not only to the naked eye, but on microscopic examination; while in the second liver, although the atrophy is extreme, so that the weight is little more than one-half that of the first, the tissue is extremely soft and friable, and there is no evidence of any increase of the fibrous tissue, either to the naked eye, or on microscopic examination. If the increased density and apparent increase of fibrous tissue in the former case be due merely to the disappearance of a portion of the secreting tissue, it would be difficult to account for the fact that in the second case, although the atrophy is much greater than in the first, the consistence of the organ is much less than in health and there is no apparent increase of the fibrous tissue. Both patients exhibited during life the ordinary phenomena of portal obstruction met with in cirrhosis; but there was this difference between the two, that the patient with the dense fibrous liver had led a very intemperate life, whereas there was no history of intemperance in the other patient. I am unable to throw any light on the etiology of the disease in the latter case; but the absence of a history of spirit-drinking, which is almost universal in the dense fibrous cirrhotic liver, is worth noting.

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[1] Third ed. p. 136.

[2] Archives of Med. vol. i. p. 125.

CASE CXII.—*No history of Spirit-drinking—Symptoms of Portal Obstruction—Soft, atrophied, granular Liver—Spurious Cirrhosis.*

Mary O——, aged 68, was admitted into Middlesex Hosp. on April 1, 1867. Her health through life had, on the whole, been good, except that at the age of 45 she had been confined to bed for six weeks with what she believed to have been rheumatic fever. Since then she had not suffered from either dyspnœa or palpitations; her habits had always been temperate. Her present illness commenced six weeks before admission with vomiting and purging. Everything she swallowed was rejected within ten minutes; these symptoms continued, and after three weeks it was first noticed that she was slightly jaundiced, and about same time she passed a good deal of blood from vagina.

On admission, patient was thin and very weak; she had a well-marked arcus senilis, and decided jaundice of skin, conjunctivæ, and urine. Pulse 108, very irregular; visible pulsation of many of arteries, which felt rigid and tortuous; impulse of heart strong and irregular, and cardiac dulness slightly increased, but no bellows-murmur audible. Respirations 28, rather laboured; coarse moist râles audible at bases of both lungs. Abdomen considerably distended and tympanitic, measuring 33 in. in circumference at umbilicus, but no distinct indication of fluid in peritoneum, and no enlargement of subcutaneous veins of abdomen. Considerable œdema of both lower extremities, but urine contained no albumen. Tongue moist, with a white fur. Motions passed after admission were dark brown, and contained abundance of bile.

On April 6 vomiting and diarrhœa had subsided, but there was tolerably clear evidence of fluid in peritoneum, and slight enlargement of subcutaneous abdominal veins; girth at umbilicus 35 in., but it never exceeded this.

From this date patient continued in a very low state, but without any increase of abdomen or, indeed, change of any sort, until April 30, when vomiting returned, but not diarrhœa. Patient now lost all appetite; tongue became dry and brown; and she continued to sink until death on May 13. For last twenty-four hours of life she was quite unconscious.

On post-mortem examination, both kidneys contracted and granular, with numerous cysts in cortical substance. Considerable hypertrophy of left ventricle of heart and atheroma of aorta, but valves healthy. Lungs slightly emphysematous, but otherwise normal. Peritoneum contained about a gallon of clear straw-coloured serum. Intestines had a fleshy appearance (from maceration) and there was slight ecchymosis in mucous membrane of cæcum, but in other respects they were normal. Spleen of natural size; with exception of a small fibrous tumour, uterus was healthy.

Liver extremely small, weighing only 25·5 oz. avoird., and measuring 7·75 in. from right to left, 5·5 in. antero-posteriorly in right lobe, and 4·75 in left. Its capsule was not at all thickened, and was not adherent; but outer surface coarsely nodulated and granular, exactly as in cirrhosis; margin of organ all round, but particularly in front, had a winged appearance, from total disappearance of secreting tissue between capsule on upper and under surfaces. At anterior margin of right lobe this attenuated rim was nearly an inch in width, and only about a third of an inch thick. On section of organ, no evidence of any increase of fibrous tissue; on the contrary, consistence was extremely soft; cut-surface presented a yellowish-brown colour, and a coarsely granular appearance, from aggregation of lobules into small masses; outline of individual lobules not well defined; but on microscopic examination secreting cells were found in abundance, though loaded with oil; no leucin nor tyrosin. The attenuated rim presented a smooth grey appearance on section, and was made up for most part of fibrillated tissue and vessels, with here and there a few collapsed secreting cells. Projecting from this rim were a few isolated nodules of yellowish-brown hepatic tissue, about size of peas.

Case CXIII. is an instance of chronic atrophy of the liver arising from peri-hepatitis. The appearance of the organ was similar to what is often seen in constitutional syphilis, although no evidence could be made out of the patient having had syphilis. The liver was very small, yet there was no indication of portal obstruction. But in another patient, whose body I dissected some years ago, there was a similar condition of liver with great ascites; the spleen weighed 27 oz., and for three days before death there had been severe vomiting and purging, with much blood in the vomited matter and stools.

CASE CXIII.—*Chronic Atrophy of the Liver from Peri-hepatitis—Simple Ulcers of the Stomach.*

The liver and stomach which I now show you were removed by me from the body of a woman, aged 44, who died in Middlesex hospital some years ago, under the care of Dr. Thompson. She was admitted on March 19, and died on April 15, 1861. Six months before admission she began to get thin, and to suffer from nausea and loss of appetite, and six weeks before admission pain and vomiting after food came on. The symptoms noted while the patient was under observation were great emaciation, tenderness in region of liver, dulness of which measured less than 2 in. in right mammary line, pain and vomiting after food, and constipation. There was no jaundice, no ascites, and no albumen in urine; heart's sounds were normal.

After death liver was found to weigh only 30½ oz.; it was very small, its dimensions being—extreme length 9 in., ant. post. diameter 6 in., greatest thickness 2 in. Capsule was thickened, and was connected to diaphragm and ribs by numerous fine long fibrous bands. Its outer surface was marked by extensive cicatrix-like depressions, and scattered through its substance were many firm fibroid gummata, about size of a pea, composed of fibrillated tissue with oily and granular matter. Glandular tissue which remained appeared healthy. Pyloric end of stomach was thickened and narrowed from what appeared to be cicatrices of former ulcers. Two inches from pylorus was an open ulcer, size of a threepenny-piece. Spleen not enlarged; commencing waxy disease of kidneys. A few small patches of recent lobular pneumonia in both lungs; base of right lung connected to diaphragm by firm adhesions. No cicatrices could be discovered on labia, in groins, or over tibiæ.

In Case CXIV. the atrophy of the liver appeared to be secondary to disease in the chest.[1]

Case CXIV.—*Bronchitis and Dilated Bronchi—Disease of Aortic Valves—Contracted Liver—Great Ascites.*

The liver which you see here is not much more than one-half of normal size; it weighed only 33 oz. Its outer surface is finely granular, being marked by numerous small depressions corresponding to the centre of the lobules. The capsule at many places is much thickened, and was adherent to the surrounding parts, and the fibrous tissue in the interior of the organ is increased. Before immersion in spirit, the surface on section presented a nutmeg appearance.

This liver was taken from the body of a man aged 40, who was a patient in this (Middlesex) hospital from June 27 to July 16, 1860, and again from November 13, 1860, until his death on January 5, 1861. He had been in habit of drinking spirits, but not in excess. His illness commenced about a year before death with cough, dyspnœa, and other signs of bronchitis. After three months his legs began to swell, and subsequently his abdomen, but at time of his first admission anasarca of legs was comparatively slight, although abdomen was enormously distended from ascites. The patient suffered much from pain below right ribs; hepatic dulness was diminished; there was no albumen in urine, but there was a diastolic blowing murmur at base of heart.

Bronchial tubes after death were found to be much thickened and dilated, and pulmonary tissue at many places was in a state of fibroid degeneration. Right lung was inseparably adherent to wall of chest. Right cavities of heart dilated; aortic valves were incompetent; two of flaps were united into one, and in all of them there was a considerable amount of atheroma.

The following case was published by me some years ago, in the Pathological Transactions, vol. vii. p. 238. It was an interesting example of chronic atrophy of the liver, in conjunction with great enlargement of the spleen and leukæmia. Although the organ was described at the time as 'in an advanced stage of cirrhosis,' the firm adhesions of the liver, omentum, and spleen, the thickened capsule of the spleen and the obliteration of the cystic duct. all pointed to chronic peritonitis as the probable cause of the atrophy. There was no history of spirit-drinking. The remarkable circumstance, however, which induces me to mention the case is that on four different occasions large quantities of fluid were abstracted from the abdomen by paracentesis, and that the patient lived for nearly two years afterwards without any reaccumulation.

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[1] See also the case of Mary T——, related in Lecture XII.

Case CXV.—*Chronic Atrophy of Liver and Ascites—Paracentesis—No Accumulation after fourth Tapping—Enlarged Spleen and Leukæmia—Death from Ulceration of Mouth and Necrosis of Jaw and Vertebræ.*

The patient was a female, who had been born and had always resided in London. She had been very temperate, but always very delicate. The catamenia had not appeared until she was twenty, and although married for eleven years, she never had any children or miscarriages. In 1850, when 31 years of age, she first noticed a swelling below right ribs; but this did not give her much inconvenience till end of 1853, when whole abdomen began to enlarge, and on April 12, 1854, she was admitted into Hospital for Women in Soho Square under Dr. Tanner.

She was then suffering from symptoms of diseased liver and ascites, and abdomen measured 43¾ in. in circumference, and 18½ in. from ensiform cartilage to pubes. During stay in hospital she was treated with iodide of potassium, mercurial ointments and purgatives, and abdomen was tapped four times. On April 17, 356 fluid oz. of clear fluid were drawn off; on May 19, 400; on June 16, 431; and on July 7, 404; altogether 1,591 fluid oz. After last operation, fluid did not collect again, and patient left hospital greatly improved in health, and with abdomen of natural size.

There was no return of ascites; but some months afterwards she began to suffer from ulceration of mouth and throat, producing a very fetid discharge. Several of her teeth came out, and in October 1855 a portion of alveolar process of lower jaw exfoliated. She had also several severe attacks of epistaxis, and bleeding from gums. On March 12, 1856, she came under my care. She was then extremely weak and confined to bed. There was extensive ulceration of fauces and along margin of gums, but voice was natural. She stated that she had never suffered from syphilis, nor taken mercury internally. Whole of left side of abdomen was filled by a solid tumour, extending forwards to within 2½ in. of umbilicus, but no ascites; hepatic dulness diminished, not exceeding 2½ in. in right mammary line.

The ulceration of mouth rapidly extended. More teeth and pieces of bone came away from jaw; body of one of cervical vertebræ became exposed; and dysphagia was so great that at last even fluids were rejected by nares.

After death, less than a pint of clear serous fluid was found in abdominal cavity, and omentum was firmly adherent to abdominal wall. Liver very small, and weighed only 35 oz. Its outer surface firmly adherent to surrounding parts, and organ was described at time as 'in an advanced stage of cirrhosis.' There was bile in gall-ducts and intestines, but gall-bladder contained none; cystic duct obliterated, and vessels and duct in portal fissure passed through a quantity of firm fibrous tissue. Gall-bladder collapsed; its lining membrane perfectly white and encrusted with small fragments of earthy matter, some of which were firmly adherent; this consisted mainly of carbonate of lime; it effervesced with acids, and microscopic examination showed it to consist of rounded crystalline particles, varying in size from $\frac{1}{400}$ of an inch to an almost infinite degree of minuteness; some of them seemed to be made up of radiating acicular crystals cohering in the centre (fig. 28).

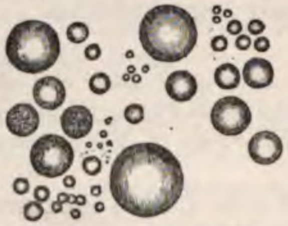

Fig. 28. Minute crystalline masses of carbonate of lime from gall-bladder in Case CXV., magnified 180 diameters.

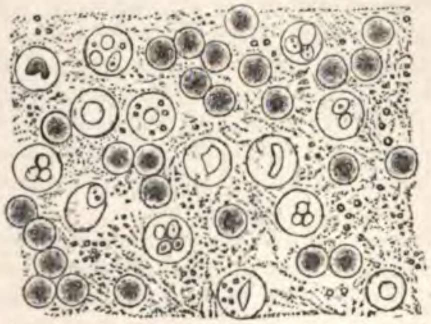

Fig. 29. Microscopic appearances of the blood in Case CXV., magnified 400 diameters.

Spleen weighed 68½ oz. and measured 11¾ in. by 4 in. Its capsule was much thickened, indurated, and adherent to surrounding parts; splenic tissue firm and of a reddish-brown colour, mottled with numerous lighter specks like sago-grains. The blood from all parts of body exhibited appearances described by Virchow in cases of leukæmia. There was a great increase of colourless corpuscles, which, on addition of weak acetic acid, presented a single, double, treble, or quadruple nucleus. Some of these nuclei were crescentic, and many of them appeared depressed in centre like small red corpuscles. Some of cells, in addition to nuclei, contained a few minute oil-globules, and there was also a considerable quantity of free granular matter and oil-globules. The red corpuscles showed no unusual tendency to collect in rolls, as in some cases of leukæmia (see fig. 29).

Heart, lungs, and kidneys healthy.

LECTURE IX.

JAUNDICE.

DEFINITION—RECOGNITION OF CAUSES—SPURIOUS JAUNDICE—PHENOMENA OF JAUNDICE: 1. LOCALITY &c.; 2. SECRETIONS; 3. BITTER TASTE; 4. DERANGEMENTS OF DIGESTION; 5. PRURITUS; 6. CUTANEOUS ERUPTIONS; 7. TEMPERATURE; 8. PULSE; 9. HÆMORRHAGES; 10. GENERAL DEBILITY AND ANÆMIA; 11. YELLOW VISION; 12. CEREBRAL SYMPTOMS—THEORY OF JAUNDICE.

GENTLEMEN,—Jaundice may be defined as a yellowness of the integuments and conjunctivæ, and of the tissues and secretions generally, from impregnation with bile-pigment. The word jaundice in fact is derived from the French *jaune*, yellow. The technical name *icterus* is less appropriate, being the Greek word for the golden thrush, a bird with golden plumage, the sight of which by a jaundiced person was believed by the ancients to be death to the bird, but recovery to the patient.

Few morbid symptoms are due to such multifarious causes as jaundice, and there are none as to which it is more difficult or important to determine the cause. It is too much the custom for even medical men to be satisfied with the fact that a patient has got 'jaundice,' and to administer remedies which are supposed to exercise some special action on the liver, without taking much trouble to investigate further; and yet the prognosis and the whole treatment of the case ought to depend, not on the jaundice, but on its cause. The difficulty of the subject is further increased by the fact, that even those who have devoted most attention to the subject are not at one as to the mode of production of jaundice in many cases. I shall endeavour in these lectures to explain to you the various causes of jaundice the mode in which they operate, and the means of distinguishing them. But in the first place it will be well that I should refer to instances of what may be called *spurious* jaundice, and describe to you certain phenomena connected with jaundice irrespectively of its cause.

SPURIOUS JAUNDICE.

In the first place, then, you must be quite certain that you have to deal with jaundice, before proceeding to investigate its cause. As a rule there is no great difficulty in the diagnosis; you have only to look at the patient to know what is the matter with him. There are, however, certain conditions which are apt to be mistaken for the slighter forms of jaundice, and it is very necessary that you should keep in remembrance these sources of error.

1. First, there is the greenish-yellow colour of countenance observed in the anæmic state known as *chlorosis*, which is due to a morbid state of the blood. This is distinguished by—

a. A pearly whiteness of the conjunctivæ and pallor of the lips and tongue.

b. Other symptoms of anæmia, such as a feeble pulse, a venous hum in the neck, and a systolic bellows-murmur at the base of the heart without any other indication of disease of the heart.

c. In females, amenorrhœa, or some other indication of uterine derangement.

d. Absence of bile-pigment from the urine.

2. There is a peculiar greyish-yellow or lemon-coloured waxen appearance of skin characteristic of organic visceral disease, and especially of cancer. This is distinguished from jaundice by—

a. The absence of any yellow tint of the conjunctivæ.

b. The absence of bile-pigment from the urine.

c. The presence of other symptoms or local signs of visceral disease, or, in the case of cancer, of the cancerous cachexia.

3. A dusky yellowish colour of the surface is not unfrequently developed in persons who have suffered long or often from malarious fevers, and sometimes also in those whose systems have been poisoned by lead, or who are the subjects of granular kidneys. This condition is to be recognised by—

a. The absence of any yellow tint of the conjunctivæ.

b. The absence of bile-pigment from the urine.

c. The fact of the individual having suffered from malarious fevers, or lived in a malarious country.

d. Exposure to the poison of lead, with a blue line along the margin of the gums, or a history of lead-colic or palsy.

e. The other symptoms of granular kidney, viz.:—copious urine of low specific gravity, containing from time to time a small quantity of albumen, hypertrophy of the left ventricle of the heart independent of valvular disease, visible arteries, &c.

4. A yellowish colour of the conjunctivæ may be due to subconjunctival fat. This is distinguished by—

a. The yellow tint not being uniform.

b. The absence of jaundice of the skin, or of bile pigment in the urine.

5. In a large number of the cases of so-called jaundice of new-born children (*icterus neonatorum*), the yellow colour which appears on the third or fourth day after birth is not due to jaundice, but is merely the result of changes in the blood in the over-congested skin, the vivid redness of the new-born babe fading as bruises fade, through shades of yellow into the genuine flesh colour.[1] Young infants, however, are liable to real jaundice, to which I shall refer hereafter. The spurious affection differs from this in—

a. The conjunctivæ being of the natural colour.

b. The urine being free from bile-pigment.

c. The gradual fading of the yellow colour of the skin after a few days.

The child being quite well, and the bowels acting properly.

6. The bronzing of Addison's disease is not likely to be mistaken for jaundice. It differs—

a. In the browner or more dusky character of the discoloration, and in the fact of its being darker at certain parts, such as the face, neck, hands, areolæ of the nipples, axillæ, penis, scrotum, &c.

b. In the presence of other symptoms of Addison's disease, and particularly of extreme anæmia and vomiting.

c. In the whiteness of the conjunctivæ.

d. In the absence of bile-pigment from the urine.

7. Persons who have been much in hot climates or exposed to the sun may have a permanent bronzed appearance of the face, which is distinguished from jaundice by—

a. The skin of the chest and other parts of the body having a natural tint.

b. Whiteness of the conjunctivæ.

c. Absence of bile-pigment from the urine.

8. Other pigments in the urine may give to this secretion a colour which may be mistaken for that of bile-pigment, such

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[1] See West, Dis. of Children, 5th ed. 1865, p. 601.

for instance, as those which are common in diseases interfering with the respiratory functions. But bile-pigment can always be recognised by the urine staining the linen yellow, and still better by testing with nitric acid. If you pour a small quantity of urine containing bile on a white plate, or on a sheet of writing-paper, and carefully allow a drop or two of nitric acid to fall upon it, an immediate play of colours will be produced around the spot where the acid falls, passing from brown through green, blue, violet, and red, into a dirty yellow.[1]

9. Lastly, those of you who may enter the public services ought to remember that jaundice has been successfully feigned by soldiers and sailors desirous of obtaining a discharge. The yellow colour of the skin has been simulated by painting it with infusions of saffron, turmeric, rhubarb, broom-flowers, or soot; while the colour of the urine has been heightened by taking rhubarb or santonin.[2] But in feigned jaundice you will find—

a. That the conjunctivæ are white.

b. That bile-pigment cannot be detected in the urine by the nitric-acid test; and,

c. That soap and water, or better still a weak solution of chloride of lime will remove the yellow colour from the skin.

d. If the urine be coloured by the use of santonin or rhubarb, it will be rendered blood-red by the caustic alkalies or their carbonates.

I will now call your attention to—

CERTAIN PHENOMENA AND CONCURRENT SYMPTOMS OF JAUNDICE IRRESPECTIVE OF ITS CAUSE.

1. The *Locality and Intensity* of the jaundice. Most of the organs and tissues of the body become impregnated with bile-pigment. This first accumulates in the blood, and the jaundiced tint penetrates almost every part of the body that is permeated

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[1] According to Frerichs this reaction may fail in consequence of the bile-pigment in the urine having undergone some transformation, in cases where the other symptoms of jaundice are undoubted. When this is the case, the urine is at one time of a brown or brownish-red colour, and becomes red on the addition of nitric acid; at another time it is of a deep red, which is converted by nitric acid into a dark bluish-red. (Dis. of Liver, Syd. Soc. Ed. i. 100.) I have made a similar observation in rare cases where jaundice has resulted from a blood-poison, and I have frequently found the urine to present these characters where there has been no jaundice, but obvious derangement of function, or alteration of structure, of the liver.

[2] Gavin, on Feigned and Fictitious Diseases, 1843, p. 389.

by blood—even the brain, the bones, and the fœtus in utero. The mucous membranes, however, are but slightly coloured, although the tongue is often distinctly yellow. The tissue of the brain and spinal cord also does not become impregnated with bile-pigment; the yellow colour which they present on section being due to the exudation of jaundiced serum from the cut ends of the vessels. Lastly, in cases of even intense jaundice, it is remarkable, from what we know of the laws of osmotic diffusion, that the humors of the eye and the crystalline lens remain free from any trace of bile-pigment: by some observers they have been found to present a yellow tinge, but the occurrence is certainly exceptional.[1]

The intensity of the jaundice varies in the different tissues of the body. When the jaundice depends on obstruction of the common bile-duct, the liver itself is the organ that is most deeply coloured; it often presents a deep olive hue. But when there is no impediment to the flow of bile into the bowel, the liver may not be more jaundiced than other parts.

Next to the liver, the skin is the tissue which becomes most jaundiced; but before it becomes affected a yellow tint is usually observed in the conjunctivæ. There must, so to speak, be a certain concentration of bile-pigment to produce a yellow colour of the skin; in the slighter and more temporary cases the conjunctivæ only may be affected. Although it has been shown by experiment upon animals, that after the passage of bile through the ducts has been arrested several days may elapse before the conjunctivæ become jaundiced, yet in the human subject twenty-four hours usually suffice for both skin and conjunctivæ to become yellow.

The colour of the skin varies from a pale sulphur or lemon-yellow, through a citron-yellow, to a deep olive or bronzed hue. The tint varies according to the cause and the duration of the disease. When the cause is obstruction of the bile-duct it is light at first, and increases in depth the longer the disease lasts. When the jaundice is independent of obstruction to the flow of bile, the colour is rarely very deep at any time, and yet these are often the most serious cases. Instances occur where the jaundice is of a greenish or almost black hue, owing to the bile-pigment which is absorbed being vitiated and dark, or to the visage being already livid from imperfect arterialisation of the blood, the green colour being a result of the mingling of the blueness of lividity with the natural yellowness of jaundice; in either case the prognosis is unfavourable. The colour also varies with the age, the natural complexion, and the amount of fat in the individual. It is deeper in the old, the wrinkled, and the dark-complexioned, than in young persons of fair complexion, and with plenty of fat. Again, in the same person, without any change in the cause, the intensity of the colour may vary from day to day, according to the diet, the amount of bile secreted by the liver and the rapidity with which it is transformed in the blood, and the activity of the bowels and kidneys. Lastly, it is important to remember in reference to treatment, that the colour often remains in the skin for some time after the cause of the jaundice has been removed, and that then its departure may be expedited by diaphoretics and warm-baths.

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[1] See Moxon, Lancet, 1873, i. 130.

2. *The Secretions* are tinged with bile-pigment, but some much more so than others. This is notably the case with the urine, by which the greater part of the bile-pigment in jaundice is eliminated from the body, and which acquires a saffron-yellow, greenish-brown, or brownish-black hue, according to the amount of pigment which it contains. The urine usually becomes yellow before there is any yellow tint of the skin, or even of the conjunctivæ, and it may happen, when the cause of the jaundice is temporary, that the whole of the pigment is eliminated by the urine, without any jaundice appearing in the skin. On the other hand, when once the skin becomes yellow, it may remain so for some time after its cause has been removed, and after bile-pigment has quite or nearly disappeared from the urine. The bile-acids have also been found in the urine of some cases of jaundice, but in cases of long standing they usually disappear.

The precipitates which fall from jaundiced urine often contain angular granules of brownish-black pigment, as well as renal epithelium and casts of the renal tubes. Tube-casts, without albuminuria, according to Dr. James Finlayson,[1] are extremely common, and appear to be due to the jaundice itself, and not to the special diseases producing it.

Other secretions in cases of jaundice contain bile-pigment, as well as the urine.

The cutaneous glands usually eliminate the pigment, and sometimes in such quantity as to stain the linen yellow, but the amount discharged in this way is small when compared with that which escapes through the kidneys.

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[1] Brit. and For. Med. Chir. Rev. Jan. 1876.

Dr. Bright[1] and others have recorded instances where the secretion of the mammary glands has been found tinged with bile-pigment, but cases of this sort are not common; while Heberden knew a woman with deep jaundice suckle her infant for six weeks without imparting to it a yellow colour, or injuring its health. Still rarer instances have been noticed where the saliva, or the tears, have been similarly affected. It is not a little remarkable, however, that, notwithstanding statements of a contrary nature which have been made by Fourcroy,[2] and Dr. Osborne of Dublin,[3] bile-pigment is not eliminated in cases of jaundice by the mucous membrane of the respiratory passages, or of the digestive tube. This is a matter of some practical importance, for, were the fact otherwise, the stools might contain bile-pigment even when there was complete obstruction of the gall-duct. But when either of these mucous membranes is inflamed and separates an albuminous or fibrinous exudation from the blood, the altered secretions may contain bile-pigment. Thus when pneumonia coexists with jaundice, there is often bile-pigment in the sputa, which may be distinguished by the nitric acid test from the greenish or yellow colour often presented by pneumonic sputa owing to changes in the blood-pigment independent of bile. Indeed, in cases of jaundice, bile-pigment may be detected in inflammatory exudations, as in the serum of a blister, before it appears in either the skin or even in the urine. It is probable that those rare cases where the saliva has been noticed to be yellow admit of a similar explanation. In the cases recorded by Huxham[4] and Budd,[5] which have been so often referred to, there was mercurial salivation, in which condition the saliva is not normal, but contains much albumen.

3. A *bitter taste* is not unfrequently complained of by persons who are the subjects of jaundice. Sometimes this appears to be due to eructations from the stomach of bilious matter, but when the bile-duct is obstructed this of course is impossible. It then probably denotes the presence in the blood of the biliary acids, for bile-pigment is tasteless, while taurocholic acid is intensely bitter. That the bitter taste is not due to the presence

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[1] Guy's Hosp. Rep. 1st Ser. i. 623. See also Budd, Dis. of Liver, 3rd ed. p. 470.

[2] Frerichs, op. cit. i. 103.

[3] Dublin Journal of Med. Feb. 1853.

[4] Op. Physico-medica, tom. iii. p. 12.

[5] Budd, op. cit. p. 469.

of bile-pigment in the blood is shown by the fact, that a similar taste is constantly complained of by persons who have hepatic derangement without jaundice.

4. *Derangements of Digestion.*—The chief derangements of digestion resulting from the absence of bile from the intestines are flatulence, constipation, and an altered character of the motions. Bile has powerful antiseptic properties, and consequently, when it is absent, the intestinal contents undergo decomposition, gases accumulate in the bowels and cause tympanitic distension of the abdomen, and the motions acquire a putrid odour. Owing to the absence of bile-pigment also the motions present a pale drab or clay colour. Bile appears also to be the natural stimulant of the peristaltic action of the gut, and consequently when the supply is cut off the bowels are usually constipated. On the other hand, the putrid fæces sometimes irritate the bowel and excite diarrhœa. The putridity and paleness of the motions and the constipation are confined to those cases where there is complete obstruction of the ducts. When the ducts are free, or where the obstruction is incomplete, and when bile still enters the bowel, the motions may be but little altered and may be voided regularly.

When bile does not enter the bowel, the digestion of fat is interfered with. Jaundiced patients dislike fat and do not assimilate it, the fatty matter in the ingesta being discharged with the fæces. This is still more remarkably the case when the pancreatic secretion is also prevented entering the bowel, but it was long since shown by Drs. Bright and Owen Rees[1] that in most cases of very obstinate jaundice, when there is complete obstruction of the bile-duct, an unusual quantity of fat may be detected in the stools. In protracted obstruction of the bile-duct there is also, as has been shown by Dr. Wickham Legg[2] and von Wittich, a complete cessation of the glycogenic function of the liver, which would account in part for the emaciation which takes place. In all cases, therefore, of jaundice from obstruction of the duct, the nutrition of the body suffers: the emaciation may be slow, but it is usually progressive, until the fat disappears from the body, and then the weight of the body may remain stationary. Cases, it is true, have been recorded where patients have lived for several years with jaundice, and where there has been comparatively little wasting, but these cases are exceptional.

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[1] Guy's Hosp. Reports 1836, Ser. 1, vol. i. p. 610.

[2] Barth. Hosp. Rep. vol. ix. 1873, and Brit. Med. Journ. Aug. 26, 1876.

5. *Pruritus* without any eruption on the skin is often a very obstinate and distressing symptom in jaundice. It sometimes precedes the jaundice, and it is a common symptom of hepatic disorder where there is no jaundice. In two cases the late Dr. Graves observed this itchiness precede the jaundice—in one for ten days, and in the other for two months—and cease as soon as the jaundice appeared. More commonly it is first noticed at the commencement of the jaundice (Case CXXVII.); sometimes it comes and goes, and at others it persists as long as the jaundice lasts, being usually worse at night and preventing sleep. You have now an opportunity of witnessing the great distress which this symptom may occasion in the case of William M—— (Case CXXVII.). This man has had jaundice from obstruction of the common bile-duct for many months, and throughout has suffered from intense itchiness, which, notwithstanding opium, subcutaneous injections of morphia, and anodynes of every sort, has caused him wretched nights. The bicarbonate of potash is the only remedy that has appeared to give relief. Pruritus is rarely observed in jaundice independent of obstruction of the bile-duct. It is not known on what ingredient of the bile this itchiness depends, but the facts of its occasionally preceding the jaundice, and of its often occurring in hepatic disorders independently of jaundice, seem to show that it is not caused by the bile-pigment.

6. *Cutaneous Eruptions.*—Urticaria, lichen, and other cutaneous eruptions, and sometimes boils and carbuncles, are occasionally observed in connection with jaundice. Dr. Graves[1] refers to eight or nine cases where persons suffering from acute rheumatism became suddenly jaundiced from the supervention of 'hepatitis' (congestion of liver?) and where the jaundice was followed by urticaria. I have not myself observed this sequence of disease, although a patient now under my care (See Lect. XI.) has been suffering from congestion of the liver with jaundice, which appeared shortly after recovery from an attack of acute rheumatism and pericarditis. Here, however, there has been no urticaria.

I must here call your attention to a very remarkable condition of the skin, named *Vitiligoidea* or *Xanthelasma*, which is now and then observed in connection with jaundice, and to which I have adverted in a former lecture (p. 247). The condition was first described by the late Dr. Addison and by Sir W. Gull, in a joint paper in the Guy's Hospital Reports,[1] and many interesting examples of the disease have of late years been exhibited to the Pathological Society.[2] The disease presents itself in two forms, either independently or in combination. In one (Vitiligoidea plana), the skin of the eyelids, of the palms of the hands and of the flexures of the fingers, and the membrane of the gums present opaque white patches, with the surface and edges slightly raised, and contrasting strongly with the surrounding jaundiced (or in the case of the gums, red) surface. These patches are not at all indurated, but their sensibility is increased ; on close examination the cuticle over them is found to be healthy, and the appearance is due to a deposit of oil in the substance of the cutis, most abundant in the neighbourhood of the hair-follicles (see p. 251). The other (Vitiligoidea tuberosa) consists of scattered tubercles of various sizes, some as large as a pea, together with shining colourless papules. The larger tubercles are tense and shining, and not unlike Molluscum ; but when punctured they give out nothing but blood, and on microscopic examination they have been found to consist of tough fibrous deposit in the true skin, infiltrated with an opalescent fluid containing fat granules. They are of a yellowish colour, mottled with a deepish rose tint, and with small capillary veins here and there ramifying over them, and they are accompanied by a moderate degree of irritation, so that their apices often appear rubbed and inflamed. They are most numerous on the face and ears, on the outside and back of the forearms, and especially about the elbows and knees, where they are often confluent. I have already pointed out to you that xanthelasma may occur in protracted jaundice from almost any cause, and that it is also observed independently of jaundice.[3]

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[1] Clinical Lect. on the Practice of Medicine, 2nd ed. vol. i. p. 446.

7. *The Temperature* of the body in jaundice dependent upon obstruction of the bile-duct, provided there be no concurrent cause of fever, is usually slightly below the normal standard,

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[1] Guy's Hosp. Rep. 2nd Ser. vol. vii. 1851, p. 265.

[2] A case is also reported by Dr. Pavy in the Proceedings of the Roy. Med. and Chir. Soc., June 12, 1866. See also a memoir by Mr. Hutchinson, Med. Chir. Trans. 1871, vol. liv. p. 171.

[3] It is worth mentioning that, in two cases at least, a similar eruption has been observed where there has been no jaundice, but where there has been diabetes, an observation of considerable interest when it is remembered how intimately connected the liver is with the pathology of diabetes. (See Addison and Gull, in Guy's Hosp. Rep. 2nd Ser. vol. ii. p. 268 ; and Bristowe, in Path. Trans. vol. xvii. p. 414.)

this reduction of heat being due to the impaired activity of the chemical processes which go on in the liver. (See Lect. XIV.).

8. *Slowness of Pulse.*—A common result of non-febrile jaundice is to retard the action of the heart and diminish arterial tension. The pulse may fall to 50, 40, or even 20, and sometimes it is also irregular. This slowness of pulse is particularly noticeable when the patient is recumbent; when he stands, the circulation is quickened. It is also accelerated when there is pyrexia in addition to the jaundice; but when fever precedes, the pulse usually falls on the supervention of the jaundice. Hence in jaundice the frequency of the pulse is a less reliable indication of fever than under ordinary circumstances, and we must trust mainly to the temperature. Slowness of the pulse is not an invariable symptom in jaundice; it is most common in simple or catarrhal jaundice, although not restricted to this form. It has not yet been explained why it is present in some cases and absent in others. The natural explanation would be that it is due to one particular ingredient of the bile, which does not exist in the blood in all cases of jaundice. Now some experiments of Röhrig,[1] continued by those of Dr. Wickham Legg,[2] and Messrs. Feltz and Ritter,[3] of Nancy, upon animals, have shown that the biliary acid salts exercise a specific paralysing action upon the ganglia of the heart and retard its action, while bile-pigment has no such effect.[3] Slowness of pulse, therefore, in jaundice may indicate the presence in the blood of unchanged biliary acids, although to this view it has been objected that, notwithstanding their diffusibility, bile-acids cannot be discovered in the urine in any form of jaundice. It may be added that I have repeatedly known the pulse sink to 36 or 40, in cases of hepatic derangement where there was no jaundice.

9. *Hæmorrhages.*—In all cases where jaundice lasts a long time, the blood becomes impoverished by a diminution in the proportion of red corpuscles and fibrin, and as a result of this there is sometimes developed a tendency to hæmorrhages from the various mucous membranes. In cases of protracted jaundice from mechanical obstruction, the immediate cause of death is not unfrequently copious hæmorrhage from the stomach or bowels. This tendency to hæmorrhage, it is true, is particularly observed in conjunction with cerebral symptoms and other

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[1] Archiv für Heilk. Aug. 1863, p. 385.

[2] Proc. Roy. Soc. 1876, No. 169.

[3] Lancet, June 24, 1876.

indications of blood-poisoning, in cases of jaundice where there is no obstruction of the bile-duct; but it also occurs in cases of mechanical jaundice of long standing, when the secreting tissue of the organ has in a great measure disappeared. I have already (pp. 264 and 280) called your attention to the frequency of hæmorrhages in cases of acute atrophy and cirrhosis of the liver, where there is no impediment to the flow of bile into the bowels.

10. *General Debility and Anæmia.*—The impaired nutrition and impoverished blood usually induce a condition of general debility and exhaustion, associated with hypochondriasis and irritability of the temper. In protracted cases there is a great diminution in the number of blood-corpuscles and corresponding anæmia.

11. *Xanthopsy, or Yellow Vision.*—In some cases of jaundice all white objects appear to the patient yellow. The symptom is extremely rare; Frerichs and other experienced observers have never met with it. There is some difference of opinion as to the mode of production of this yellow vision. If the humors of the eye became impregnated with bile-pigment, yellow vision might be expected in all cases of jaundice; but I have already told you that even in intense jaundice, the rule is, that these humors and the crystalline lens are not in the slightest degree tinged. It has yet to be shown whether xanthopsy is peculiar to those rare cases in which the humors become tinged with bile. In several instances where xanthopsy was present Sir Thomas Watson noted a distended condition of the vessels of the conjunctivæ, and he refers to a case of Dr. Elliotson's, where yellow vision was limited to one eye covered with varicose vessels; he accordingly concludes that it is only when the vessels of the eye are large enough to transmit blood-globules, that they give passage to the bile-pigment which tinges the humors of the eye.[1] Now you will remember that there is considerable enlargement of the conjunctival vessels in the case of William M—— (Case CXXVII.), who for a short time had yellow vision, but that, although he no longer has xanthopsy, the enlargement of the vessels remains. The fact that the yellow vision often intermits without any change in the jaundice, and that it is frequently absent when there is intense jaundice of the cornea and of the other tissues of the eye (Frerichs), and the statement that it has been met with in typhus fever where there has been no jaundice, and that it is sometimes associated

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[1] Lect. on the Principles and Practice of Physic, 5th ed. vol. ii. p. 677.

with other derangements of vision such as night-blindness, have led many to regard it as a purely nervous symptom. The ophthalmoscope may possibly clear up the existing doubts respecting this curious symptom. It may be mentioned that after the use of santonin yellow vision is sometimes observed, which ceases as soon as the colouring matter is eliminated from the blood by the kidneys; it would be interesting to know if the santonin enters the humors of the eye.

12. *Cerebral Symptoms*, such as acute delirium, stupor, coma, convulsions, muscular tremors, subsultus, carphology, paralysis of the sphincters, a dry brown tongue, and other indications of the 'typhoid state' occasionally supervene in cases of jaundice. They are most common in cases where there is no obstruction of the ducts, but they may also occur in cases of long-standing obstruction, where all or a greater part of the secreting tissue has been destroyed. Different opinions have been held as to their cause. In exceptional cases they are due to inflammation of the membranes covering the upper surface of the brain; but as a rule, after death no lesion of the brain or of its membranes is found to account for them, and they must therefore be due to some alteration of the blood. They are commonly attributed to poisoning of the blood with bile, and many experiments have been performed on animals to show that bile, or the biliary acids,[1] is a deadly poison. That dogs should die after injection into the cellular tissue of the bile of other dogs is not extraordinary, and admits of another explanation than that of the essential elements of bile being a poison. The injection of mucus from another dog would probably produce a like result, and all bile contains mucus.

Pure bile, from which the mucus has been removed, has been repeatedly injected into the large veins of dogs by Frerichs and other observers, without any cerebral symptoms or bad results ensuing, except that death in some instances has been caused by the entrance of air into the veins.[2] The operation has been even repeatedly performed on the same animal without any lasting injury. But it is scarcely necessary to turn to experimental enquiriés on the lower animals for evidence on the matter, and in all these experiments there are sources of fallacy. You have had abundant proof in the wards, that the blood and tissues of the human subject may be saturated with bile for months (and I may add, for years), without any cerebral symptoms resulting. Those of you who have witnessed the case of William M—— (Case CXXVII.), who for many months has had permanent closure of the bile-duct, will find it difficult to believe that bile or any of its ingredients is a deadly poison.

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[1] See, for example, Harley on Jaundice, p. 39.

[2] Diseases of Liver, Syd. Soc. Ed. vol. i. p. 395.

An American physician, Dr. Austin Flint, jun., has endeavoured to show that the cerebral symptoms in jaundice are due to the retention of cholesterin in the blood, or to what he has designated *cholestearæmia.* Cholesterin is a crystalline fatty matter, and is one of the constituents of the complex substance, bile. Dr. Flint regards it as an excrementitious product of nervous tissue, the elimination of which from the body is one of the functions of the liver.[1] Arrived in the bowel, the cholesterin, according to him, is converted into *stercorin*, and therefore it is not found in the fæces, but when retained in the blood he believes it to be a poison like urea. But if the non-excretion of all the elements of bile does not give rise to cerebral symptoms, it is difficult to understand how these symptoms can result from the retention of cholesterin alone. In cases, for instance, of permanent closure of the duct, cholesterin is not discharged from the liver into the bowel, nor does it accumulate in the biliary passages, nor does it produce cerebral symptoms if it be retained in the blood. There are moreover cases on record where there has been permanent closure of the bile-duct, followed by almost entire destruction of the secreting tissue of the liver, and where in consequence this organ has been incapable of eliminating any of the elements of the bile which may be preformed in the blood, and yet where no cerebral symptoms have been noticed. Arguing from such cases Dr. Budd contends that when cerebral symptoms occur in jaundice, they are due to some peculiarly noxious matter which is evolved, *in consequence of decomposition*, in the lobular substance of the liver.[2] No such noxious matter, however, has yet been discovered.

The cerebral symptoms in jaundice resemble those produced by many known blood-poisons, but the poison is more probably generated in the blood, and throughout the tissues generally, than in the liver in particular. The liver is not merely an excretory organ, but unquestionably exercises an important influence on the metamorphoses of matter constantly taking place in the blood and tissues, and although the precise nature of these changes is insufficiently known, there are reasons for believing that the liver is instrumental in the production of urea and uric acid. When, for example, the functions of the liver are arrested, one result is that urea is not elaborated, but substances such as leucin and tyrosin, and perhaps others with which we are as yet imperfectly acquainted, of a composition intermediate between urea and the protein compound (see p. 263), are developed; while the materials which ought to be eliminated from the body as urea and uric acid accumulate in the blood. In acute yellow atrophy and in the yellow fever of the tropics the occurrence of cerebral symptoms is marked by an extraordinary diminution of urea in the urine. The pathology in fact of the cerebral symptoms in jaundice is probably very similar to what I have endeavoured to prove to you is the pathology of the typhoid state in all diseases.[1] But to this subject we shall return after considering the theory of jaundice.

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[1] American Journ. of Med. Science, Oct. 1862; and Récherches Expér. sur une nouvelle fonction du Foie, Paris, 1868.

[2] Dis. of Liver, 3rd edit. pp. 270, 475.

THEORY OF JAUNDICE.

All cases of jaundice may be referred to one of two classes, viz.—

I. Cases in which there is a mechanical impediment to the flow of bile into the duodenum, and where the bile is in consequence retained in the biliary passages, and thence absorbed into the blood.

II. Cases in which there is no impediment to the escape of bile from the liver.

These two forms of jaundice have long been recognised; but great differences of opinion have been held, and still exist, as to the mode of production of the jaundice in the second class of cases, and yet these are the cases which are, perhaps, the common in practice.

When any obstruction exists to the flow of the
the hepatic or the common duct, the way in w
arises is sufficiently clear. The bile-ducts and
become distended with bile, which is abso
by the lymphatics and the veins. This wa

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[1] See Abstract of a Clinical Lecture on the P
Typhoid State in different Diseases. Brit. Med. Jo

at the beginning of this century by the experiments of Dr. Saunders,[1] which have since been confirmed by other observers. If a ligature be applied to the hepatic duct of a dog, and the animal be killed after two hours, the lymphatics in the walls of the bile-ducts, which are very numerous, are seen to be distended with a yellow fluid; the fluid in the thoracic duct is also yellow, and so likewise are the intervening lymphatic glands. In patients also who die of obstruction of the bile-duct, the lymphatics of the liver are often found to contain bile. On the other hand, the serum of blood taken from the hepatic vein two hours after ligature of the common duct is found to contain much more bile-pigment than that of blood taken from the jugular vein.[2] This preponderance of bile-pigment in the blood of the hepatic veins over that of the general circulation shows that bile, in cases of obstruction of the gall-duct, is also directly absorbed by the veins. Indeed, as we shall presently find, there is reason to believe that even when there is no obstruction, bile is constantly passing from the gall-bladder and biliary passages into the circulation, in virtue of the law of diffusion of fluids through animal membranes. Under ordinary circumstances jaundice does not result, because the bile is at once transformed in the blood, and in its turn influences the metamorphosis of other matters, the products of which metamorphosis are eliminated by the urine. But in the distension of the biliary passages consequent on obstruction, the pressure upon, and the extent of, the diffusing surface are increased, and consequently more bile enters the blood than can undergo the metamorphosis necessary for its elimination by the urine. Even in obstruction, however, the intensity of the jaundice (or the amount of unchanged bile accumulated in the blood) will vary with the amount of bile secreted by the liver, the rapidity with which it is eliminated by the kidneys, and the activity of oxydation going on in the blood.

But in a large proportion of cases there is no mechanical impediment to the escape of bile from the liver, and then an explanation of the jaundice is less obvious. Boerhaave and Morgagni long ago suggested that the jaundice in these cases was the result of a *suspended secretion*. They maintained that the function of the liver was merely to separate the elements of bile which were already formed in the blood, and that when anything interfered with this function of the liver, the blood retained the ingredients of the bile, and the result was jaundice. Although this view was strenuously opposed in this country at the beginning of the century by Dr. Saunders, who contended that 'in every case of jaundice bile must be secreted and carried into the blood-vessels,'[1] it is the view which is generally received at the present day. Dr. Budd, for instance, in his treatise on Diseases of the Liver remarks, 'in these cases the most obvious explanation of the facts is, that the biliary pigment exists in the blood, and that in consequence of defective action of the secreting cells, it is not eliminated as it should be in the liver.'[2] It is right to add, however, that Dr. Budd makes a special exception with regard to the biliary acids.[3] 'The most skilful chemists,' he says, 'who have recently analysed the portal blood, have failed to detect the biliary acids in it, and have come to the conclusion that these, at least, are formed in the liver.'[4] This view, that the liver manufactures the bile-acids, while it merely excretes the bile-pigment, is also adopted by Dr. G. Harley in his essay on Jaundice.[5]

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[1] Treatise on the Structure, Economy, and Diseases of the Liver, and on Bile and Biliary Concretion, 3rd ed. 1803.

[2] Dr. Legg states that he has recently repeated this experiment in the dog without success. St. Bartholomew's Hospital Reports, vol. ix. 1873.

It seems to me, however, that there are weighty objections to the view, that even the bile-pigment is formed in the blood

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[1] Op. cit. p. 107.

[2] Op. cit. p. 468.

[3] Bile is a very complex substance. Its composition, according to Gorup-Besanez, is as follows:—

Water	822·7	to	908·1
Solid matter	177·3	,,	91·3
Bile-acid salts	107·9	,,	56·5
Fat and cholesterin	47·3	,,	30·9
Mucus and pigment	23·9	,,	14·5
Ash	10·8	,,	6·3

Two acids have been found in the bile, which have been named by Lehmann glycocholic and taurocholic acid. According to this chemist these acids are formed by the conjugation of cholic acid with glycin (gelatin-sugar) and taurin respectively, and they are united in the bile with soda as a base. The composition of glycocholic acid is $C_{26}H_{43}NO_6$, and that of taurocholic acid, $C_{26}H_{45}NO_7S$. Two modifications of bile-pigment have been found, viz. a brown pigment, named cholepyrrhin, bilifulvin, or bilirubin, and a green pigment, biliverdin; cholepyrrhin is convertible into biliverdin. Very little is known of the chemical nature of these substances; there are probably other modifications of the pigmentary matter, which, as well as those mentioned, are the products of the transformation or oxydation of one primitive substance. See also Lecture XIV.

[4] Op. cit. pp. 40, 467.

[5] Jaundice, its Pathology and Treatment, by G. Harley, M.D. Lond. 1863.

and merely excreted by the liver, some of which may be mentioned.

1. Although bile-pigment appears to be derived from the colouring matters of the blood and may be produced from this by the action of chemical reagents, or may even be developed in extravasations as a pathological product,[1] it has not yet been satisfactorily shown that bile-pigment, as such, exists ready formed in the blood of persons who have not jaundice. Frerichs denies that it ever has. Lehmann, who has investigated with great care the changes which the blood undergoes in passing through the liver, has never been able to detect the colouring matter of bile in portal blood, and infers that this as well as the bile-acids must be formed in the liver itself.[2] The blood of the hepatic artery has been examined with a like result. It is obvious that if bile-pigment exist in healthy blood at all, its quantity must be very minute; and when it is remembered that the daily quantity of bile manufactured in the liver is about two pints, and yet that jaundice is not a normal condition, it seems impossible that all the bile-pigment secreted by the liver can be formed in the blood; and it is not probable that part is formed in the blood, and part by the liver.

2. The discovery by a few observers of a small quantity of bile-pigment in what appeared normal blood does not prove that it was formed in the blood, for it is conceivable that it may have been formed in the liver and been then absorbed. It is probable, indeed, as I shall endeavour to prove to you presently, that bile-pigment is constantly being absorbed into the blood, becoming altered in the act of absorption or immediately after; and if this be so, it is quite possible that a trace of it should occasionally remain unaltered in the blood without giving rise to obvious jaundice.

3. If the constituents of bile are formed in the blood, intense jaundice ought at once to follow the extirpation of the liver in any of the lower animals, in like manner as urea accumulates in the blood after removal of the kidneys. But so far from this being the case, Müller, Kunde, Lehmann, and Moleschott have

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[1] See Virchow's Cellular Pathology, Engl. Transl. pp. 128, 145, and Kuhne, Lehrbuch der physiologischen Chemie, Leipzig, 1866, p. 89. Analyses of the bile-pigments, which have been communicated to the Royal Society by Dr. Thudichum, tend to show that they have no relation to hæmatin, as was formerly supposed. (Proc. Roy. Soc. 1867, vol. xvi. p. 220.)

[2] Physiological Chemistry, Dr. Day's transl. vol. ii. p. 87.

repeatedly extirpated the liver of frogs, and have invariably failed to find a trace either of the biliary acids, or of the colouring matter of the bile, in the blood, the urine, or the muscular tissue.[1]

4. It often happens that from various diseases, such as fatty and waxy degeneration, cancer, and cirrhosis, the secreting tissue of the liver in the human subject is for the most part or entirely destroyed, bile is no longer secreted, and yet no jaundice results. Several cases of this sort are referred to by Haspel, where the gall-bladder after death contained only a little white mucus.[2] Frerichs also records a case of fatty liver where the contents of the bowels were pale, the gall-bladder empty, and the biliary ducts coated with a greyish mucus, notwithstanding which the skin was of a chalky paleness, and the urine contained no bile-pigment.[3] Similar observations have been made by Dr. Budd, in cases of waxy disease and cancer of the liver,[4] and several instances of a like nature have come under my own notice. If bile be formed in the circulating blood, it is difficult to account for what becomes of it in these cases.

These considerations make it very doubtful if any form of jaundice can with propriety be attributed to a suppression of the hepatic functions. It remains then to be considered if any more satisfactory explanation can be offered of those cases of jaundice in which there is no impediment to the flow of bile from the liver into the duodenum.

A solution of the difficulty has been proposed by Professor Frerichs of Berlin. According to this distinguished observer, a large proportion of the colourless biliary acids formed in the liver is either directly taken up by the blood in the hepatic vein, or is absorbed from the bowel. Under ordinary circumstances, these biliary acids become oxydised and assist in forming the large quantity of taurin found in healthy lung and the pigments voided in the urine; but these normal metamorphoses are liable to interruption by nervous agencies, or by poisons in the blood, and then the bile-acids, not being sufficiently oxydised, are converted into bile-pigment in the blood, and the result is jaundice.[5] This view has been supported by two experiments intended to show: 1. That bile-pigment can be obtained artificially from the bile-acids, by the action of concentrated sulphuric acid;

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[1] Carpenter's Human Physiology, 7th ed. p. 434.

[2] Malad. d'Algérie, i. 262.

[3] Op. cit. Eng. Ed. i. 83.

[4] Op. cit. pp. 329, 411.

[5] Op. cit. vol. i. pp. 89, 394.

and, 2. That, colourless biliary acids, when injected into the veins of dogs, are converted in the blood of these animals into bile-pigment. These experimental results, as well as the conclusions drawn from them, are still the subject of much discussion. They have been controverted by Kuhne,[1] Hoppe, Harley,[2] &c., but supported by Staedeler,[3] Neukomm,[3] Folwarczny,[3] Röhrig,[4] &c. It has been contended on the one hand, that the biliary acids in these experiments are decomposed in the blood; and on the other, that in whatever manner they find their way into the blood, they are excreted unchanged by the kidneys. The majority of observers seem to concur with Frerichs; and his view is confirmed by the circumstance, that of the large quantity of bile-acids secreted by the human liver and subsequently absorbed none appears in the urine. But the decision of the question at issue is not of material importance for explaining those cases of jaundice in which there is no impediment to the escape of bile from the liver, inasmuch as there are grounds for believing that not only in jaundice but in health, a portion of the bile-pigment, as well as of the bile-acids formed in the liver, is absorbed into the blood.[5]

1. The quantity of bile-pigment discharged with the fæces is but a fraction of what is calculated to be secreted by the liver.[6] Speaking of the principal constituents of bile, Dr. Carpenter[7] remarks: 'the further we descend in the intestinal canal, the less of them do we meet with:' and again he says: 'of the bile which is poured into the alimentary canal, a large part is certainly reabsorbed, its constituents being destined to undergo oxydation and be eliminated, for the most part by the respiratory processes: and it is probably from this reabsorbed portion of the bile that the sulphur of the urine is derived.' According to Dr. Bence Jones, also, 'the colouring matter (of the

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[1] Virchow's Archives, vol. xiv. pts. 3 and 4, Sept. 1858, and Beale's Archives of Medicine, vol. i. p. 342.

[2] Pathology and Treatment of Jaundice, 1863.

[3] See my Preface to the English Edition of Frerichs on the Liver, pp. xv, xvi.

[4] Archiv für Heilkunde, Aug. 1863, p. 385.

[5] It may be thought improbable that the liver should secrete from the portal vein a material which is afterwards to be absorbed by the branches of the same vessel. But it has, perhaps, been too readily assumed from the comparatively large size of the vena portæ that it furnishes all the materials of bile (see Lecture XIV.)

[6] For further evidence on this subject the reader is referred to the author's Croonian Lectures on Functional Derangements of the Liver. (Lecture XIV. in this Volume.)

[7] Carpenter's Princ. of Hum. Physiology, 5th ed. pp. 102, 353, 374.

bile) undergoes changes in the intestines, and some of it most probably in health is carried into the blood and textures, and is finally removed in the colouring matter of the urine.'[1] It is the knowledge of this circumstance that offers the only satisfactory explanation of the remarkable discrepancy of opinion in the profession respecting mercury and other substances, which are supposed to exercise some specific effect upon the liver in stimulating it to an increased secretion of bile. The practical physician gives a dose of calomel, finds the quantity of bile in the motions greatly increased, and argues that the liver has been stimulated to an increased secretion; but the physiologist ties the common bile-duct, makes a fistulous opening into the gall-bladder, and then finds that calomel has no effect on, or even diminishes, the amount of bile that drains away through the fistula.[2] Mercury and allied purgatives probably produce bilious stools by irritating the upper part of the bowel, and sweeping on the bile before there is time for its absorption; irritating articles of diet will often produce precisely the same effect. Calomel is of unquestioned utility in congestion of liver, but if it acted, as is usually argued, by stimulating the liver to increased secretion, it might be expected to increase the congestion rather than diminish it. It is possible, however, that the irritation of the duodenum by purgatives may be reflected to the gall-bladder, and cause it to contract, and that the evacuation of this viscus may account in part for the increased quantity of bile in the stools.

2. From what is now known of the diffusibility of fluids through animal membranes, it is impossible to conceive bile long in contact with the lining membrane of the gall-bladder, bile-ducts, and intestine, without a portion of it (including the dissolved pigment) passing into the blood. A circulation in fact is constantly taking place between the fluid contents of the bowel and the blood, the existence of which till within the last few years was quite unknown, and which even now is too little heeded.[3] 'It is now known,' says Dr. Parkes, in his Gulstonian Lectures on Pyrexia, 'that in varying degrees, there is a constant transit

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[1] St. George's Hospital Reports, vol. i. p. 192.

[2] On the Influence of Mercurial Preparations on the Secretion of Bile, by George Scott, M.D. Beale's Archives of Medicine, vol. i. 209.

[3] For instance, the purging of cholera is probably the result of some stoppage in this intestinal circulation—of a diminished power of absorption, rather than of an increased exhalation from the mucous membrane of the bowel. Numerous facts render it probable that in cholera the power of absorption is greatly impaired or abolished.

of fluid from the blood into the alimentary canal, and as rapid reabsorption. The amount thus poured out and absorbed in twenty-four hours is almost incredible, and of itself constitutes a secondary or intermediate circulation never dreamt of by Harvey. The amount of gastric juice alone, passing into the stomach in a day and then reabsorbed, amounted in the case lately examined by Grünewaldt,[1] to nearly 23 imperial pints. If we put it at 12 pints we shall certainly be within the mark. The pancreas, according to Kroeger, furnishes 12½ pints in twenty-four hours, while the salivary glands pour out at least 3 pints in the same time. The amount of the bile is probably over 2 pints. The amount given out by the intestinal mucous membrane cannot be guessed at, but must be enormous. Altogether the amount of fluid effused into the alimentary canal in twenty-four hours amounts to much more than the whole amount of blood in the body; in other words, every portion of the blood may, and possibly does, pass several times into the alimentary canal in twenty-four hours. The effect of this continual outpouring is supposed to be to aid metamorphosis; the same substance, more or less changed, seems to be thrown out and reabsorbed until it be adapted for the repair of tissue or become effete.'[2]

It is in the course of this osmotic circulation that the constituents of bile are taken up into the blood, becoming transformed in the process of absorption into products which are eliminated by the lungs and kidneys,[3] while at the same time they assist in the assimilation of the nutritive materials derived from the food. And here we have an explanation of those cases of jaundice where there is no impediment to the flow of bile from the liver. Under normal conditions, the whole of the bile that is absorbed is at once transformed, so that neither bile-acids nor bile-pigment can be discovered in the blood or in the urine, and there is no jaundice. But in certain morbid states the absorbed bile does not undergo the normal metamorphoses; it circulates in the blood and stains the skin and other tissues. The morbid states, which, so far as we know, conduce mainly to this result, are precisely those in which we might expect abnormal blood-metamorphosis, viz.—

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[1] An account of this case, abstracted by me from Grünewaldt's Latin Memoir, will be found in Beale's Archives of Medicine, vol. i. p. 270. C. M.

[2] Med. Times and Gazette, April 7, 1855, p. 333.

[3] In various diseased conditions of the liver, even when there is no jaundice, or bile-pigment in the urine, this fluid is rendered very dark, sometimes almost black, by boiling and adding nitric acid.

1. Certain poisons, such as those of yellow fever, relapsing fever, pyæmia, and more rarely those of remittent fever, typhus, scarlatina; also snake-poison, chloroform, &c.

2. Nervous influences, such as a sudden fright, violent rage, great or protracted anxiety, and concussion of the brain.

3. A deficient supply of oxygen, as happens in certain cases of pneumonia in persons living in confined and crowded dwellings.

4. An excessive secretion of bile, especially when conjoined with constipation. In this case, unless the bile be removed by purging, the quantity absorbed may be too great to undergo the normal metamorphosis, and the presence in the blood of the untransformed bile causes jaundice.

According to this view, the only pathological difference between jaundice from obstruction and jaundice independent of obstruction of the common bile-duct is that in the former case none of the bile secreted by the liver can escape from the body by the fæces, and consequently all that is secreted, after the gall-bladder and biliary passages are fully distended, is absorbed into the blood, the quantity thus absorbed being far too great to undergo the normal metamorphoses; while in the latter case bile passes into, and is discharged from, the bowel, as usual, but that which is absorbed, which in quantity may not exceed that which is absorbed in health, remains unchanged in the blood.[1] As might have been expected, the jaundice in the former case is much more intense than in the latter, although where an obstruction of the bile-duct has lasted long the jaundice often becomes paler, not from removal or diminution of the obstruction, but from the secreting tissue of the liver being destroyed and comparatively little bile being secreted; while in cases where there is no obstruction of the bile-duct, the intensity of the jaundice will vary according to the amount of bile which is absorbed and the degree of derangement of the blood-metamorphoses.

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[1] According to Dr. Moxon and Dr. Hilton Fagge, 'this theory, that jaundice is in all cases due to reabsorption, is entirely inconsistent with the fact that in jaundice the biliary passages are almost always found to contain, not bile, but an almost colourless mucus. This is the case, not only in acute yellow atrophy of the liver, but also when the ducts are permanently obstructed by cancerous growths, gall-stones, &c.' (Trans. Path. Soc. 1873, vol. xxiv. p. 129; and Guy's Hosp. Rep. 1875, vol. xx.) This argument has been met by Dr. Wickham Legg, who writes as follows: 'The presence of a colourless fluid in the gall-bladder and the bile-ducts was formerly looked upon as evidence that the liver had ceased to secrete bile. Indeed, a recent writer (W. Moxon) is still plainly of this opinion. But, to my mind, the evidence seems rather the contrary. It should be remembered that it was the large ducts which were seen to be filled with this colourless fluid, and that nothing is said of the state of the smaller ducts, of the interlobular and capillary ducts. These continue to receive the bile poured into them by the liver-cells, but the bile does not reach the large ducts because the small ducts are shut off from the large, either by plugs of this tenacious fluid or by gravel. In a case which I recently examined at St. Bartholomew's, the large ducts were perfectly colourless; but, by gently pressing the liver, a yellow fluid could be made to issue from the small ducts. Also, by careful dissection, the small ducts could be seen to be stained yellow.' (Brit. Med. Journ. 1874.)

Lastly, we may enquire what explanation the theory of jaundice now advanced gives of the cerebral symptoms met with in certain cases and already referred to (p. 322). From what has been stated it is very probable that the entrance of bile into the blood is necessary to perfect those metamorphoses from which materials for the urinary solids are derived. At all events, this seems certain, that when the secreting tissue of the liver is destroyed, as in acute atrophy and in certain cases of long-standing obstruction of the bile-duct, these metamorphoses are imperfectly executed. Urea is not formed in sufficient quantity, and substances such as leucin and tyrosin, of intermediate composition between it and the protein compounds (see p. 263), accumulate in the blood and tissues and appear in the urine. These are the circumstances under which cerebral symptoms occur in cases of so-called 'suppression of bile.' The mere presence of bile in the blood, as I have already shown you (p. 322), will not account for them, and indeed in those cases where cerebral symptoms are most apt to supervene, the jaundice as a rule is less intense than it often is when they are absent.

The detailed consideration of the various causes of jaundice, and of the means of distinguishing them, we must reserve for subsequent lectures.

LECTURE X.

JAUNDICE.

CLASSIFICATION OF CAUSES OF JAUNDICE—JAUNDICE FROM OBSTRUCTION OF THE BILE-DUCT.

GENTLEMEN,—After the preliminary remarks on the subject of jaundice made in the preceding lecture, we may now proceed to consider its different causes, and the means of distinguishing them.

All cases of jaundice, as I have told you, may be conveniently grouped under the two heads of—

A. Jaundice resulting from obstruction of the Common Bile-Duct; and,

B. Jaundice independent of any obstruction of the Bile-Duct.

The numerous causes comprised under each of these heads may be seen from this Table:

TABULAR VIEW OF THE CAUSES OF JAUNDICE.

A. JAUNDICE FROM MECHANICAL OBSTRUCTION OF THE BILE-DUCT.

I. OBSTRUCTION BY FOREIGN BODIES WITHIN THE DUCT.
  1. Gall-stones and inspissated bile.
  2. Hydatids and Distomata.
  3. Foreign bodies from the intestines.

II. OBSTRUCTION BY INFLAMMATORY TUMEFACTION OF THE DUODENUM, OR OF THE LINING MEMBRANE OF THE DUCT, WITH EXUDATION INTO ITS INTERIOR.

TABULAR VIEW—*continued.*

III. OBSTRUCTION BY STRICTURE OR OBLITERATION OF THE DUCT.

1. Congenital deficiency or obstruction of the duct.
2. Stricture from peri-hepatitis.
3. Closure of orifice of duct in consequence of an ulcer in the duodenum.
4. Stricture from cicatrisation of ulcers in the bile-ducts.
5. Spasmodic stricture?

IV. OBSTRUCTION BY TUMOURS CLOSING THE ORIFICE OF THE DUCT OR GROWING IN ITS INTERIOR.

V. OBSTRUCTION BY PRESSURE ON THE DUCT FROM WITHOUT, BY

1. Tumours projecting from the liver itself.
2. Enlarged glands in the fissure of the liver.
3. Tumour of the stomach.
4. Tumour of the pancreas.
5. Tumour of the kidney.
6. Post-peritoneal or omental tumour.
7. An abdominal aneurism.
8. Accumulation of fæces in bowels.
9. A pregnant uterus.
10. Ovarian and uterine tumours.

B. JAUNDICE INDEPENDENT OF MECHANICAL OBSTRUCTION OF THE BILE-DUCT.

I. POISONS IN THE BLOOD INTERFERING WITH THE NORMAL METAMORPHOSIS OF BILE.

1. The Poisons of the various specific fevers:
   a. Yellow fever.—*b.* Remittent and Intermittent fevers.—*c.* Relapsing fever.—*d.* Typhus.—*e.* Enteric or Pythogenic fever.—*f.* Scarlatina.—*g.* 'Epidemic Jaundice.'
2. Animal Poisons:
   a. Pyæmia.—*b.* Snake-poison.
3. Mineral Poisons:
   a. Phosphorus.—*b.* Mercury.—*c.* Copper.—*d.* Antimony, &c.
4. Chloroform and Ether.
5. Acute Atrophy of the liver?
6. Cirrhosis and other forms of Chronic Atrophy of the liver.

TABULAR VIEW—*continued.*

II. IMPAIRED OR DERANGED INNERVATION INTERFERING WITH THE NORMAL METAMORPHOSIS OF BILE.

1. Severe mental emotions, fright, anxiety, &c.
2. Concussion of the brain.

III. DEFICIENT OXYGENATION OF THE BLOOD INTERFERING WITH THE NORMAL METAMORPHOSIS OF BILE.

IV. EXCESSIVE SECRETION OF BILE, MORE OF WHICH IS ABSORBED THAN CAN UNDERGO THE NORMAL METAMORPHOSIS.

Congestion of the Liver:

a. Mechanical.—*b.* Active.—*c.* Passive.

V. UNDUE ABSORPTION OF BILE INTO THE BLOOD FROM HABITUAL OR PROTRACTED CONSTIPATION.

I shall now endeavour to describe to you the distinguishing characters of the several forms of jaundice referred to in the Table.

A. JAUNDICE FROM MECHANICAL OBSTRUCTION OF THE BILE-DUCT.

I. OBSTRUCTION BY FOREIGN BODIES WITHIN THE DUCT.

1. *Gall-stones or Inspissated Bile.*

Gall-stones are among the most common causes of Jaundice from Obstruction. It very commonly happens that the gall-bladder is found full of concretions after death, as in the specimens I show you here, and yet that there have been no symptoms during life to lead to any suspicion of their existence. Gall-stones only produce jaundice and other symptoms when they enter the bile-duct, and the most characteristic symptoms are those which are produced by the *passage of the concretions along the duct.* In most cases where there are the symptoms of gall-stones, there is a distinct concretion or calculus; but similar symptoms occasionally result from what is called inspissated bile, or from a gritty condition of the bile. It is not often that you have an opportunity of proving this by post-mortem examination, although it is a fact of some clinical importance, inasmuch as it accounts for some of those cases where there have been the symptoms of gall-stones, but where none could be found in the stools. You will find, however, a case related by Dr. Handfield Jones in the fifth volume of the Pathological Transactions (p. 150), where a woman died of universal jaundice a short time after having fractured her thigh by a fall, and where the lower end of the common duct was found quite plugged up with 'a sandy matter consisting of biliary pigment.' You know also that all the phenomena of nephritic colic may be produced by the passage of lithic acid sand.

The jaundice resulting from gall-stones, as a rule, is not difficult to diagnose.

1. The passage of a gall-stone along the common duct, unless it be a very small one, gives rise to the pain known as biliary colic. The patient is often forewarned of the attack by a feeling of nausea with much flatulence, an unusual nervous excitability, yawning, or shivering. The attack usually comes on shortly after the principal meal,[1] or after some severe muscular exertion or shaking of the body. Very often the patient is suddenly seized with violent pain, but more commonly the pain is moderate at its onset and gradually increases in severity. The pain starts from the epigastrium and radiates to both hypochondria, to the spine, to the right shoulder, to both shoulders, to the left alone, or to the neck, but never downwards.[2] It is usually of two sorts, a dull aching pain which is constant; and an acute agonising pain, which comes and goes in paroxysms, and which is described as of a boring, tearing, burning, or constricting character. The latter is often so excruciating that the patient will bend himself double, with his chin resting on his bended knees, and constantly shift his position with the object of obtaining relief. Women who have borne many children will tell you that the pains of childbirth are nothing in comparison to those of biliary colic. Now and then, in nervous persons, the pain excites epileptiform convulsions. The paroxysms, if frequent and protracted, induce great lassitude and exhaustion, the face being pale, the pulse slow, and the whole body covered with a cold sweat; occasionally there is profound collapse, which in rare cases has been fatal; and in three

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[1] Cullen's definition of biliary colic was: 'Icterus, cum dolore in regione epigastrica, acuto, post pastum aucto, et cum dejectione concretionum biliosarum.'

[2] I cannot confirm Trousseau's statement, that, although it more generally ascends, 'the pain goes down into the abdomen, in some cases simulating nephritic colic.' Clin. Lect. Syd. Soc. Ed., iv. 233.

instances I have known an attack terminate in fatal coma.[1] At its onset the pain may be relieved by pressure; but after it has lasted long there is almost always some tenderness over the fundus of the gall-bladder, which persists for some time after the paroxysm has subsided and is a useful symptom in diagnosis. Occasionally this tenderness is acute from the gall-bladder having become inflamed. Trousseau has called attention to the circumstance that an attack of hepatic colic is sometimes followed by an intercostal neuralgia, distinguished by tenderness over some of the dorsal spines.[2] Lastly, it is stated that biliary calculi are in rare instances found in the stools, without there having been any symptoms of biliary colic.[3] Dr. Fagge also relates the case of a man who died of hernia, and who had previously had jaundice unattended with pain; his gall-bladder contained numerous gall-stones, and the common duct was dilated so as to admit the finger.[4]

2. Rigors, often severe, recurring at irregular intervals, but sometimes periodically almost with the exactness of an ague, are not uncommon in severe and protracted cases, and are believed to depend on over-distension of the gall-bladder and bile-ducts. In reference to this symptom a remarkable specimen is preserved in the Pathological Series of the Royal College of Surgeons.[5] It is that of a large oval calculus fitted tightly into the end of the common bile-duct, a portion of it projecting through the dilated orifice of the duct into the duodenum. The patient from whose body the preparation was obtained was a very large woman, aged 70, who for nearly six months had been subject to spasmodic pains of the stomach, coming on with shiverings like an ague-fit, which lasted for half an hour or an hour, and were succeeded by unusual heat. It was only during the last month of life that vomiting and jaundice had set in. Three days before death she was seized with an unusually severe attack of shivering and pain, which continued, with

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[1] The first case was a lady aged 76, whose urine contained much albumen. The second was a lady aged 84, whose urine also contained albumen; she recovered from the first attack of coma; but six months after she had a recurrence of biliary colic, followed by coma which was fatal. The third case was a Hindoo lady, aged 50, whose urine could not be obtained.

[2] Op. cit. vol. i. p. 482.

[3] A case in point is related by Dr. Sénac of Vichy. Du Traitement des Coliques hépatiques, Paris, 1870, p. 46.

[4] Guy's Hosp. Rep. 1875, vol. xx.

[5] The preparation is from the collection of Mr. John Howship, and is numbered 1459.

scarcely any remission, until death. In Case CXVII., although there were no decided rigors, there was a remarkable periodicity in the attacks.

3. Vomiting accompanies the paroxysms in most cases and is often frequent and severe, the patient rejecting all food that may be in the stomach and bringing up large quantities of acid fluid. Bilious vomiting indicates that the common bile-duct is still free. The act of retching is usually followed by a temporary alleviation of the pain. Very often there is frequent hiccup.

4. After these symptoms have lasted from twelve to twenty-four hours, jaundice usually appears, and if the obstruction of the bile-duct continue for a few days the jaundice becomes intense, the urine being loaded with bile-pigment, giving it a deep mahogany hue, and the fæces containing none. At the commencement of the paroxysm, before the appearance of jaundice, the patient often passes large quantities of limpid nervous urine, and occasionally similar urine is voided on some particular occasion during the persistence of jaundice, although that which is passed immediately before and after contains abundance of bile. The occurrence of jaundice may be said to clench the diagnosis of the nature of abdominal colic, but it is not invariably present although the colic be hepatic. If the stone finds its way into the bowels within twenty-four hours, or if it does not get beyond the cystic duct, or if its form is so angular as not entirely to block the flow of bile, it is quite possible to have biliary colic without jaundice. Many patients suffer from repeated attacks of what is put down as gastralgia or cramp in the stomach, without any jaundice, but whose subsequent history leaves little doubt that the attacks have been due to gall-stones. In these cases the diagnosis is sometimes assisted by observing that the attack of pain is followed by the slightest yellow tint of the conjunctivæ, or by the presence of bile-pigment in the urine. In Case CXVII. the patient suffered from severe biliary colic daily for four months before jaundice appeared, the stone, from its great size, being all this time detained in the cystic duct; and Trousseau mentions a case in which biliary colic occurred repeatedly during four years, and then for the first time jaundice occurred. There is also good authority for the statement that small biliary calculi have been found in the stools of individuals who have never had jaundice.[1]

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[1] Trousseau, op. cit. iv. 236.

but such cases are certainly exceptional; and I am unable to understand the statement of Wolff that 25 out of 45 patients observed by him passed through the whole train of symptoms of biliary colic without jaundice, the concretions being found in the evacuations.[1] The duration of the jaundice will vary with the number and size of the stones, but usually it does not last longer than from a few days to a few weeks. It is not very often that a gall-stone leads to permanent jaundice, for if it succeed in escaping from the cystic duct, where its presence will not cause jaundice, it will usually find its way through the larger common duct. Cases, however, are occasionally met with, such as Case CXVII. where permanent and even fatal jaundice has been caused by the impaction of a gall-stone.[2] But although the jaundice of gall-stones be in most cases of temporary duration, it has this peculiarity, that it is liable to recur with the other symptoms already referred to at irregular intervals, owing to some of the concretions not escaping from the gall-bladder during the first attack, or to fresh ones forming in the place of those which have been discharged. The diagnosis then is often materially assisted by the patient having had a similar attack on some former occasion. The very fact of a person in middle or advanced life having had several attacks of well-marked jaundice, with distinct intermissions, would point to gall-stones as the probable cause. At the same time you must remember that when a large calculus has forced its way through the natural channels of the bile, they will remain permanently dilated, and smaller stones may be afterwards voided without either jaundice or pain. According to Sir Thomas Watson, there are persons who get rid of scores of stones in this way during the course of their lives.

5. The jaundice from gall-stones is usually unaccompanied by fever; there is no increase of temperature, and the pulse is oftener below the normal standard of frequency than above it. During the paroxysms of pain, however, increased frequency of pulse and a temporary rise of temperature, even when the patient is shivering and the extremities feel cold, are not uncommon; also, if the pressure of the calculus has induced inflam-

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[1] Virchow's Archives, vol. xx. pt. 2, 1861.

[2] In the Pathological Transactions, a case of fatal jaundice is recorded by Dr. Handfield Jones (vol. v. p. 146), where the hepatic and common ducts of the liver were obstructed by large calculi; and another is reported by Dr. J. Wale Hicks, where the cystic duct and part of the common duct were occupied by a large gall-stone, which also projected into the gall-bladder (vol. xv. p. 126).

mation or ulceration of the biliary passages, there may be persistent or intermittent pyrexia after the pain has ceased, and under these circumstances fresh attacks of pain are often followed by a temporary pyrexia ending in perspiration. Now and then it happens that a gall-stone in one of the biliary ducts excites paroxysms of intermittent fever, with little or no pain. These paroxysms may be more or less periodic, and may extend over several months without necessarily indicating pyæmic hepatitis (see p. 166), the patient ultimately recovering. Charcot[1] has attributed these attacks to a septic poison, the product of chemical changes in the bile within the dilated and inflamed ducts; but more probably they are due to the simple irritation of the stone, and are analogous to the febrile paroxysms resulting from the passage of a catheter along the urethra.

After the paroxysms of biliary colic are over, the urine usually throws down copious sediments of lithic acid or lithates.

6. If the obstruction of the bile-duct persists for several days there will often be found a slight and uniform enlargement of the liver, with a tender pyriform tumour corresponding to the gall-bladder, resulting from the great dilatation of the biliary passages by the accumulated bile, as I have explained to you in a former lecture (p 160).

7. The diagnosis of gall-stones will be assisted by remembering the circumstances under which they are most likely to be met with.

a. Sex.—They are more common in females than in males (3 to 2).

b. They are chiefly met with in persons of middle and advanced life. Of 395 cases collected by Hein, only 15 were under twenty-five years of age, and only three under twenty. The liability to the formation of gall-stones probably increases with advancing age, but not the risk of biliary colic. According to Dr. Sénac[2] of Vichy, who has paid particular attention to this subject, hepatic colic most frequently commences about the age of thirty-five, and comparatively rarely after fifty. You must remember, however, that gall-stones are occasionally met with in early life. In a previous lecture I have detailed to you a case where they occurred at the age of twenty-three (Case LXXI. p. 173): and rare cases have been observed of gall-stones in children[1] and even in young infants. In the first volume of the Northern Journal of Medicine (p. 240) you will find a case recorded where fatal jaundice in a new-born babe was due to obstruction of the bile-duct by 'an indurated cord-like plug of inspissated bile;' and many years ago Lieutaud reported the case of an infant, 25 days old, in whom a gall-stone completely obstructed the orifices of the hepatic and pancreatic ducts.[2]

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[1] Le Progrès Méd. Aug. 1876.

[2] Du Traitement des Coliques hépatiques, Paris, 1870, p, 56.

c. *Climate.*—In warm climates, notwithstanding the liability to hepatic derangements, gall-stones are very rare.

d. *Habits.*—Gall-stones are particularly common in persons of stout habit, who consume large quantities of rich saccharine and greasy food and alcoholic fluids, and who at the same time lead sedentary lives.

e. *Social Position.*—From what has been stated it follows that gall-stones are much more common in the middle and upper classes than among the labouring population and the poor.

f. *Associated Diseases.*—In a very large proportion of cases of gall-stones there will be found to be a history in the patient or in his family of gout, asthma, urinary gravel, neuralgia, migraine, or urticaria. This is a point upon which I have insisted in my Croonian Lectures on Functional Derangements of the Liver, and in which the extensive experience of Dr. Sénac is quite in accordance with mine.

g. *Hereditary.*—From the frequent concurrence of gall-stones with gout and allied maladies, it is not to be wondered at that gall-stones are in many instances hereditary. We constantly meet with several members of the same family who have suffered from biliary colic.

h. *Exciting Causes of Biliary Colic.*—Gall-stones being already in the gall-bladder, an attack of biliary colic is often determined by an overloaded stomach, menstruation, a fit of indigestion, a sudden strain, a fall, driving over a rough road, or some severe mental emotion.

8. But the most conclusive proofs of jaundice being due to gall-stones is finding the concretion in the fæces. It is not only satisfactory to the patient to see the stones, but their appearance is often of some use in prognosis. If one large globular concretion have been passed, it is very possible that the patient may not be further troubled: but if the stone be marked by several flat surfaces or facets, such as I show you here, the

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[1] Trousseau mentions a case in a girl aged nine.

[2] Mém. de l'Acad. Roy. de Méd., 1847 xiii. 264.

probability is that there are several, or many, more. But even when all the symptoms above described have been present in a marked degree, you may fail in finding a gall-stone in the fæces. This may be due to the concretion becoming disintegrated in the bowel, or to its slipping back into the gall-bladder instead of into the duodenum, or to the obstruction of the duct having been caused by inspissated gritty bile, rather than by a distinct concretion; but too often it is the result of a faulty method of search. The common belief is that gall-stones are lighter than water, and that therefore if water be poured on the fæces, any gall-stones present will float; but Sir Thomas Watson, who recommended this method in the earlier editions of his Lectures, added :—'I never but once succeeded in thus catching a concretion in the evacuations of a patient, where symptoms had led me to search for it.' In a later edition, however, he says that three other patients, taught how to search for them, had detected in the alvine discharges this palpable source and explanation of their previous sufferings.[1] The truth is that most gall-stones, before they are dried, are heavier than water, in which they will not float; and accordingly the plan which you have seen followed in the wards, and which is the only reliable one, is to pass the whole evacuation from the bowels through muslin or a sieve. Gall-stones also are often not found in the fæces owing to the search for them not being maintained sufficiently long. Trousseau relates the case of a patient who never passed the stone from the bowel until three, four, or five days after the termination of the attack of biliary colic.[2] I may add that a German physician, Wolff, who took the pains to examine the fæces sometimes for months after an attack of biliary colic, never failed to find gall-stones in one of 45 cases of biliary colic occurring in his practice during a period of forty-three years.[3]

2. *Hydatids, Distomata, and Lumbrici in the Bile-Ducts.*

Hydatid tumours of the liver, as I have already told you (see p. 66), occasionally burst into the bile-duct. If the tumour contain no secondary cysts, its fluid contents may be discharged through the bile-duct into the duodenum, and the patient may get well without any marked symptoms. But in most cases there are secondary cysts which enter and obstruct the bile-duct, and produce all the symptoms of jaundice from an impediment to the flow of bile. In a former lecture I have related to you cases where this occurred (Cases XXXII. to XXXIV. p. 112). The passage moreover of the hydatid cysts along the bile-duct may give rise to severe paroxysms of pain, rigors, and vomiting, and in fact to all the phenomena of the biliary colic resulting from gall-stones. This happened, you will remember, in a marked manner in Case XXXIV. From gall-stones, however, the case would be distinguished—

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[1] Lect. on Practice of Physic, 2nd ed. ii. p. 527, and 3rd ed. ii. p. 555.

[2] Op. cit. vol. iv. p. 228.

[3] Virchow's Archives, vol. xx. pt. 1, 1861.

1. By there being the physical signs of hydatid enlargement of the liver already described to you (p. 55), with perhaps a subsidence of the swelling on the occurrence of pain.

2. By there being in most cases symptoms of persistent fever, a quick pulse and elevated temperature, in addition to those of biliary colic. When the hydatid bursts into the bile-duct, not only do vesicles enter the duct, but bile enters the hydatid, and the consequence is that this inflames and suppurates and causes fever. Where, however, as in Case XXXIV., vesicles continue to pass along the bile-duct long after the bursting of the tumour, there may be biliary colic without fever.

3. The diagnosis will be complete on detecting hydatid vesicles in the alvine evacuations, as was done in Case XXXIV. (p. 116).

In those rare instances to which I have already (p. 66) directed your attention, where a hydatid tumour appears to be developed in the first instance in the bile-duct, its diagnosis will probably be impossible.

In cases of extreme rarity the *Distoma hepaticum*, or liver-fluke, which is so common in the livers of sheep, has been found in the biliary passages of the human subject, but its presence does not appear necessarily to obstruct the duct and cause jaundice. In Davaine's great work on Entozoa, the case is related of a girl, aged 8, who died in the hospital at Milan of diarrhœa, marasmus, and convulsions, and on opening whose body there was found a pouch containing five distomata near the termination of the common bile-duct. This patient had suffered from all the symptoms of biliary colic, but had no jaundice.[1] Some years ago a patient died in this (Middlesex) hospital, whose gall-bladder was found to contain a fluke. The lining membrane of the gall-bladder was perfectly white, but Dr. Budd, who relates the case, does not state whether there was any obstruction of the common duct or jaundice.[1] The distoma in the sheep causes dilatation and catarrh of the biliary passages, with atrophy of the hepatic tissue and great anæmia, but only in rare cases jaundice. The diagnosis of distomata in the bile-ducts of the human subject could only be arrived at in the event of any of the parasites being ejected by vomiting or in the stools.

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[1] Traité des Entozoaires, 1860, p. 252.

There are also not a few instances where round worms have penetrated the orifice of the bile-duct and caused jaundice, with biliary colic, vomiting, and all the symptoms of gall-stones.[2] Some years ago I saw in the Museum of the General Hospital at Vienna a specimen (No. 1312) showing the common bile-duct dilated to the size of a man's thumb, and obstructed by a large mass of round worms. Several of these cases have proved fatal suddenly by convulsions. It is also worth noting that in many of these cases the bowels have contained a number of worms, and that there has been a history of worms being ejected by vomiting, or passed per anum. It is by such an occurrence alone that any diagnosis of the cause of the jaundice could be arrived at.

3. *Foreign Bodies from the Intestine.*

Foreign bodies, such as cherry-stones and currant-seeds, have been known to enter the bile-duct from the intestine, and to give rise to jaundice. But in those rare cases where this has happened, it is probable that the bile-duct has been already dilated by the passage of a gall-stone. Several curious cases are on record where the nucleus of a gall-stone has been found to be a dried-up round worm, the fragment of a distoma, a needle, or a plum-stone.

II. JAUNDICE FROM OBSTRUCTION BY INFLAMMATORY TUMEFACTION OF THE DUODENUM, OR OF THE LINING MEMBRANE OF THE BILE-DUCT, WITH EXUDATION INTO ITS INTERIOR.

When a mucous membrane inflames, it becomes swollen from the increased amount of blood in its vessels and from œdematous infiltration of the submucous tissue, while at the same time the secretion from the surface is increased in quantity and altered in quality. If these changes take place in the mucous membrane lining a narrow tube like the bile-duct, one can easily understand that the passage through it should be blocked up, and this in fact is what often happens. Catarrhal inflammation is one of the most common causes of mechanical jaundice, and is certainly the most common cause of jaundice in young persons. To this cause are referable most of the cases commonly described as 'simple jaundice.' Its symptoms and the circumstances under which it occurs have been fully described to you in a former lecture (p. 152). In a large number of cases, as I told you, the inflammation commences in the duodenum and spreads up the bile-duct, and sometimes the duodenal orifice of the duct may be found effectually blocked up by the tumid mucous membrane of the duodenum or by a plug of viscid mucus, without the inflammation having extended further up the duct.

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[1] Dis. of Liver, 3rd ed. p. 494.

[2] Frerichs, Dis. of Liver, Engl. Transl. ii. p. 482; Morehead, Dis. of India, 1st ed. 1856, ii. 155. Davaine, op. cit. p. 156; and particularly, Bonfils, 'Des Lésions et des Phénomènes pathologiques déterminés par la presence des Vers Ascarides Lumbricoïdes dans les canaux biliaires,' Archiv. Gén. de Méd., Juin, 1858, p. 661; and Vinay, 'Observ. d'Ictère generalisé tenant à la presence de Lombrics dans les voies biliaires,' Lyon Méd., 1869, i. 251.

In diagnosing the causes of jaundice, it is important to remember that inflammation of the biliary passages may be caused by gall-stones, and that thus the symptoms of these two causes of jaundice may coexist; or it is possible that inflammation of the biliary passages may be excited by gall-stones which have never produced biliary colic. In the case of J. K. (Case LXV. p. 159) you will remember that the inflammation of the biliary passages seemed to be excited by gall-stones, which were found in the gall-bladder and in the bile-ducts, and yet that the most careful enquiry failed to elicit any history of biliary colic. The paroxysmal pain is caused by the passage of the calculus *along the duct.* Concretions which never leave the gall-bladder may also excite inflammation of its lining membrane, and this may spread to the bile-ducts, but will not give rise to biliary colic.

Lastly, it must be remembered that the bile-duct may be easily closed by a catarrhal swelling, which, as in the case of œdema of the glottis, could not be demonstrated after death.

III. JAUNDICE FROM OBSTRUCTION TO THE FLOW OF BILE BY STRICTURE OR OBLITERATION OF THE BILE-DUCT.

1. *Congenital Deficiency or Obstruction of the Duct.*

I have already told you that in the majority of cases of the so-called *icterus neonatorum* the yellow colour of the skin is not jaundice at all (p. 312). At the same time infants are liable to real jaundice, which is sometimes a serious symptom. It may, as we have seen, depend upon a plugging of the duct with inspissated bile (p. 342), and then there may be some hope of the obstruction giving way and of the child recovering; or, as I shall have occasion to explain to you in another lecture, it may depend on deficient oxygenation of the blood interfering with the normal metamorphosis of bile. At other times it has a pyæmic origin, and is associated with peritonitis, or with phlebitis of the umbilical vein; and lastly it may be due to a congenital closure, obliteration, or absence of the bile-duct, no trace of it remaining except a little areolar tissue between the hepatic artery and portal vein. The gall-bladder in these cases is extremely small and collapsed, and sometimes it also is absent; but in the duodenum the opening of the pancreatic duct may be found as usual. Cases of this sort have been recorded or collected by Dr. A. D. Campbell, in the Northern Journal of Medicine for 1844, by Dr. Wilks, in the 13th volume of the Pathological Transactions (p. 119), by Dr. West in his standard work on the Diseases of Infancy and Childhood (5th ed. p. 605), and more recently by Dr. Binz of Bonn, in Virchow's Archives.[1] In not a few of the cases which have been recorded, there has been evidence of intra-uterine peri-hepatitis, and strange to say, notwithstanding the rarity of the malformation, many writers have referred to several instances of it occurring in the same family. These considerations suggest the desirability of enquiry, whether these malformations be not sometimes one of the results of peri-hepatitis from hereditary syphilis.

The following case (Case CXXI.) came under my own notice a few years ago, in the out-patient department of this (Middlesex) hospital.

Jaundice from this cause may be recognised by the following characters:—

a. The jaundice appears within a few days of birth, and gradually increases in intensity. The conjunctivæ as well as the skin are yellow.

b. The motions are white, and the urine leaves a yellow stain on the clothes.

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[1] Zur Kenntniss des tödtlichen Icterus der Neugebornen aus Obliteration der Gallengänge. Archiv f. path. Anat. und Physiol. und f. klinische Medicin, Berlin 1866, vol. xxxv. p. 360. References to several other papers on the subject will be found in Dr. West's work quoted above; also Glasgow Med. Journ. Jan. 1876, p. 11.

c. In most cases there have been observed hæmorrhages from the umbilicus (often fatal), from the bowels, beneath the skin, and in other parts of the body, as in Case CXXI.

d. At first the child may appear strong and healthy, but very soon progressive atrophy sets in, attended often with vomiting and diarrhœa, and death usually occurs within a few months of birth. In one of Dr. Campbell's cases, and in another related by Dr. West, the infant lived for six months.

2. *Stricture of the Bile-Duct from Peri-hepatitis.*

In peri-hepatitis the lymph which is thrown out becomes after a time organised, and causes thickening of the capsule and firm fibrous bands of adhesion connecting the liver to surrounding parts. Now and then it happens that the new areolar tissue, which is formed in this way in the portal fissure, exerts a constricting effect on the bile-duct and sometimes also on the portal vein, and the result is, in the one case jaundice, and in the other ascites, with the other signs of portal obstruction already referred to (see pp. 275–284). You will find a case of this sort related by Frerichs.[1] It will often be difficult to recognise during life this cause of jaundice, but the following characters may sometimes be of assistance in diagnosis:

1. A history of some of the usual causes of peri-hepatitis, such as simple ulcer of the stomach, inflammation of the right pleura, general peritonitis, other diseases of the liver, and especially constitutional syphilis.
2. An antecedent history of the symptoms of peri-hepatitis, and more particularly of acute pain and tenderness in the right hypochondrium with more or less pyrexia.
3. The concurrence of symptoms of chronic atrophy of the liver and signs of portal obstruction (see p. 277).
4. The absence of any history of biliary colic, or of any indications of cancer.
5. The fact of the jaundice, when once it appears, being permanent, not intermittent.

3. *Closure of Orifice of the Duct in consequence of an Ulcer in the Duodenum.*

This is another cause of mechanical jaundice of which the recognition during life is often very difficult. Simple ulcers, like those of the stomach, occasionally form in the duodenum and may, like them, end in hæmorrhage or perforation. Occasionally it happens that one of these ulcers is situated at the part of the duodenum corresponding to the opening of the bile-duct, and this becomes obstructed by inflammatory products which are apt to become organised, and then the obstruction is permanent. A like result may ensue from the end of the duct being involved in the cicatrix of a duodenal ulcer, as happened in the case of James B., who died in this (Middlesex) hospital (Case CXXII.). In the diagnosis of this cause of obstruction we must be guided by—

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[1] Op. cit. vol. i. p. 151.

1. The circumstance of the jaundice and other signs of obstruction of the bile-duct being preceded by the symptoms of ulcer of the duodenum, such as pain felt only two or three hours after a meal, when the food is passing from the stomach into the duodenum, with perhaps occasional attacks of sudden and profuse hæmorrhage from the stomach or bowels. These symptoms, however, are often absent in cases of ulcer of the duodenum, which may indeed run such a latent course that its existence is not suspected until the occurrence of fatal perforation.[1]

2. In any case of persistent and intense jaundice with complete disappearance of bile from the motions, the very absence of all symptoms antecedent to those of obstruction of the bile-duct, while at the same time there is no evidence of a tumour, of ascites, or of the cancerous cachexia, and no history of biliary colic, will lend some probability to the view that the cause of obstruction is a duodenal ulcer. There is, however, a source of fallacy in the fact that an ulcer of the duodenum, near the opening of the common duct, has been known to induce attacks of spasmodic abdominal pain followed by jaundice. In such a case an absolute diagnosis from gall-stones would be impossible; for although the occurrence of the paroxysms immediately after obvious errors in diet, or in conjunction with the symptoms of duodenal ulcer already referred to, might point to this as the probable cause, yet duodenal ulcer, as I have told you, is often a remarkably latent disease. Fortunately cases of this sort are so rare as not often to embarrass the diagnosis.

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[1] In the ninth volume of the Pathological Transactions (p. 197), I have recorded the case of a large finely developed man who died suddenly of peritonitis, from a perforating ulcer of the duodenum, and who up to the time of his fatal attack had enjoyed excellent health, and had never suffered from vomiting or even pain after meals. Similar cases have been related by Dr. Budd, in his Lectures on Diseases of the Stomach (p. 149), and by other writers.

IV. OBSTRUCTION BY TUMOURS CLOSING THE ORIFICE OF THE DUCT OR GROWING IN ITS INTERIOR.

The channel or opening of the bile-duct is liable to become obstructed by cancerous and other growths in the duodenum; by tumours in the pancreas, gall-bladder, or adjacent parts, penetrating the part of the duodenum where the duct opens or the duct itself in any part of its course;[1] or, in rare cases, by growths originating in the walls of the bile-ducts themselves. In Case CXXIV. there was a cancerous tumour in the head of the pancreas, but the cause of the jaundice was an independent growth in the bile-duct. Another case of jaundice from obstruction of the bile-duct by a tumour developed in its walls is recorded by Dr. Bristowe, in the ninth volume of the Pathological Transactions (p. 220). Case CXXVI. is an example of cancer of the pancreas, involving in its growth the gall-bladder and the bile-duct; and you will find two similar cases recorded by Frerichs, in which the cancer had invaded the duodenum and obstructed the orifice of the bile-duct.[2] The chief characters on which we must rely for the diagnosis of this cause of jaundice are as follows:—

1. Before the jaundice appears, the patient usually complains for some weeks, or longer, of pain, more or less severe, and sometimes lancinating, in the region of the duodenum. This pain continues after the appearance of jaundice, and is usually aggravated two or three hours after taking food.

2. In most cases there are nausea and a tendency to vomit, especially after food.

3. Hæmorrhage from the stomach or bowels is occasionally met with when there is cancerous ulceration of the duodenum.

4. In some cases a hard and tender tumour can be felt more or less distinctly, on careful examination through the abdominal parietes.

5. The jaundice, when once it appears, gradually increases in intensity and lasts till death, which, it is important to add, occurs in most cases within four or five months of the first appearance of yellowness of the skin. The very circumstance of jaundice lasting in any case longer than six months would be

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[1] In the Pathological Transactions (vol. xxiv. p. 103) Dr. Sydney Coupland has reported a case where a cancerous ulcer of the duodenum just beyond the pylorus, invaded the gall-bladder, and from this the cystic and hepatic duct, so as to cause jaundice without implicating the common bile-duct.

[2] Op. cit. vol. i. Cases VI. and VII.

carefully recorded and analysed, but you will remember that this lesion sometimes gives rise to pyæmia with multiple abscesses in the liver (see pp. 166 and 341); and, independently of pyæmia, it is probable that, as in Mr. Holmes's case above referred to, the ulceration will be ushered in with chilliness and rigors, and be accompanied by pain or uneasiness in the hepatic region, and pyrexia with great variations of temperature. From these symptoms I have diagnosed ulceration of the bile-ducts in one or two cases of jaundice, but I have had no opportunity of verifying the diagnosis.

5. *Spasmodic Stricture of the Bile-Duct.*

When the common bile-duct becomes constricted or obliterated from any of the four causes just mentioned, the jaundice is deep and permanent, there is progressive emaciation, and death sooner or later is the result. But it was formerly imagined[1] that temporary jaundice might result from spasmodic constriction of the duct, constituting what was called *icterus spasmodicus*; and indeed all cases of jaundice where no mechanical obstruction to the flow of bile could be found after death were at one time explained in this way. The contractility of the bile-ducts has been demonstrated by experiment, when they have been mechanically irritated or galvanised in an animal just dead, and it is very possible that during life the passage of irritating bile may cause spasmodic contraction of the duct with severe pain, in the same way as spasm of the bowel is believed to cause colic, and spasm of the bronchi, asthma. It is very doubtful, however, if jaundice ever results from spasmodic contraction of this sort. I have already told you (p. 314) that even mechanical obstruction of the duct takes a day or longer to produce jaundice of the integuments, and it is difficult to conceive that a spasmodic stricture, independent of any mechanical obstruction, could last sufficiently long to produce a like result. I have also explained to you how catarrhal obstruction of the bile-duct may cease with death, and how jaundice may be developed independently of any impediment to the flow of bile, so that it is unnecessary to have recourse to the theory of spasm to account for those cases where no mechanical obstruction of the bile-duct can be found after death.

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[1] See, for example, Saunders's Treatise on the Structure, Economy, and Diseases of the Liver, and on Bile and Biliary Concretions, 3rd ed. 1803, p. 100; and Sir Thomas Watson's Lectures on Medicine, 3rd ed. vol. ii. p. 557.

its channel and cause jaundice.[1] In a large proportion of the cases of cancerous or waxy liver where jaundice is observed, it is produced in this way. I may, for instance, recall to your recollection the case of Hannah C—— (Case LXXXV., p. 216), who died of cancer of the liver and ovary, and whose jaundice was due to compression of the bile-duct by a mass of enlarged glands and dense areolar tissue in the portal fissure. In cases also of primary cancer of the stomach cancerous matter is often infiltrated into the lesser omentum, and may compress the bile-duct and produce jaundice. The recognition during life of the cause of obstruction of the bile-duct in these cases must depend mainly on :—

1. The signs and symptoms of waxy disease or cancer of the liver, which I have described to you in former lectures, or of cancer of the stomach, or general tuberculosis, or lymphoma.

2. The co-existence in most cases of ascites, owing to the pressure being exerted on the portal vein as well as on the bile-duct (see Case LXXXVII., p. 219).

3. *Tumour of the Stomach.*

A cancerous tumour of the pyloric end of the stomach may cause jaundice by mere compression of the bile-duct. More commonly the duct is compressed by secondary cancerous deposits in the lesser omentum, or in the glands of the portal fissure. The cause of the jaundice in such a case may be recognised :—

1. By the fact of its being preceded and accompanied by the ordinary symptoms of cancer of the pylorus, and more particularly by pain and vomiting after food, coffee-ground vomit, and rapid emaciation.[2]

2. By the situation of the tumour, and by the fact of its being often accompanied by great dilatation of the stomach, distinguishable through the abdominal parietes.

4. *Tumour of the Pancreas.*

A tumour of the pancreas may not only, as we have seen, invade the duodenum and close the orifice of the bile-duct, or penetrate and obstruct the duct at different parts of its course; but, when large, it may compress the duct from without so as to constrict or obliterate its channel. The symptoms of this form of obstruction of the bile-duct will not differ much from those of obstruction from a cancerous tumour of the duodenum (see p. 352), viz.:—

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[1] See a case recorded by Dr. Handfield Jones in the Pathological Transactions, vol. v. p. 149.

[2] See a case reported by Dr. Bristowe, in the ninth volume of the Pathological Transactions, p. 225, Case VII.

1. Pain referred to the situation of the pancreas.
2. Nausea and tendency to vomit.
3. A distinct hard tumour often appreciable at the seat of pain.
4. Jaundice permanent until death.
5. Rapid emaciation and other indications of the cancerous cachexia.
6. The passage in some cases of a large quantity of fatty matter in the stools (see p. 317).

The phenomena arising from obstruction of the bile-duct by a cancerous tumour in the pancreas may be closely simulated by an abscess in the pancreas, secondary to a simple ulcer of the duodenum, which involves and obstructs the opening of the bile-duct in the manner already described (see p. 349). This appears to me to have been the pathology of a case recorded by Dr. G. Harley, in the thirteenth volume of the Pathological Transactions (p. 119).

5. *Tumours of the Kidneys.*

Great enlargement of the kidneys, according to Dr. Copland,[1] may cause jaundice by the pressure exerted by the tumour on the bile-duct; but jaundice from this cause must be extremely rare, as I have been unable to find a case in illustration recorded in the Pathological Transactions, or elsewhere, and I have frequently known the kidneys enormously enlarged from various causes without any jaundice resulting. In a former lecture I called your attention to a case where there was an enormous cystic tumour of the right kidney (Case VIII., p. 27), containing at least 200 oz. of fluid; and I show you here a cancerous tumour of the left kidney, from a boy aged 8, which weighed 496 oz. and filled almost the whole abdomen.[2] In neither of these cases was there jaundice. I have, however, met with more than one instance in which the right kidney was enormously enlarged from cancer, and where jaundice was induced by secondary deposits in the glands in the portal fissure.

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[1] Dictionary of Medicine, vol. ii. p. 302.

[2] The details of this case were published by Dr. Vanderbyl in the Pathological Transactions, vol. vii. p. 268.

The diagnosis of jaundice from the pressure of an enlarged kidney on the bile-duct must rest on the clinical characters of enlargement of the kidney, to which I have already referred in my first lecture (p. 14).

6. *A Retro-peritoneal or Omental Tumour.*

A tumour originating behind the peritoneum and growing forwards into the abdomen may ultimately involve and compress the bile-duct and cause jaundice; and a tumour—cancerous, colloid, or tubercular—originating in the omentum may lead to a like result.[1] The bile-ducts passing through the morbid growth are compressed and narrowed, and rendered completely impervious. These tumours are usually cancerous, and by the time that they are large enough to compress the bile-duct their existence is sufficiently obvious. The chief difficulty in diagnosis will arise in determining the place of origin of the tumour. It may be difficult, for instance, to distinguish an omental tumour in the vicinity of the liver and compressing the bile-duct from a tumour of the liver itself; and, indeed, for the solution of the question we must trust entirely to its history and mode of growth. Unfortunately, so far as prognosis and treatment are concerned, the precise locality of origin of the tumour matters little. No known treatment can prevent, or long defer, the fatal event.

7. *An Abdominal Aneurism.*

In very rare cases of aneurism of the abdominal aorta, the tumour, when very large, may compress the bile-duct and cause jaundice. Dr. Hutton, for instance, has recorded a case where the tumour reached from the crest of the ilium to the lower end of the scapula and caused jaundice. You will find it referred to in Dr. Stokes's classical work on Diseases of the Heart and Aorta.[2] But in aneurism of certain of the branches of the abdominal aorta jaundice is more common, and it is particularly liable to be produced by aneurisms of the hepatic artery. Frerichs has collected four cases[3] of aneurism of the hepatic artery from different sources, from which it would appear that although the lesion is rare, it has well-defined characters during life. These are mainly—

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[1] See a case of cancer of lesser omentum in Pathological Transactions, vol. ix. p. 225; and another of colloid of lesser omentum, ib. vol. xvii. p. 136, both recorded by Dr. Bristowe.

[2] The Diseases of the Heart and Aorta, 1854, p. 633.

[3] Frerichs, op. cit. vol. ii. p. 378. Frerichs makes five cases, but one of the five,

1. Symptoms of imperfect duodenal digestion, pain in the duodenum and its vicinity occurring two or three hours after taking food.

2. Paroxysms of acute neuralgic pain in the region of the liver, simulating the colic of gall-stones, and due, no doubt, to the pressure of the aneurism on the hepatic plexus of nerves.

3. Persistent jaundice from compression of the bile-duct.

4. Attacks of hæmatemesis and bloody stools, and great anæmia in consequence.

5. A tumour in the right hypochondrium, which may displace the liver upwards. The nature of the case would be rendered still more certain by detecting in this tumour pulsation and a single or double bellows-murmur. In a case, however, recorded by Dr. Stokes, there was no pulsation.

In three of the four cases collected by Frerichs the aneurism burst before death—twice into the abdominal cavity, and once into the gall-bladder.

Similar symptoms have been noticed in cases of aneurism of the superior mesenteric artery, although in this form of aneurism jaundice is less common, and hæmorrhages perhaps more so. There was no jaundice in the patient from whose body I removed this specimen some years ago—a man aged 42, who died in this (Middlesex) hospital on September 27, 1860, of profuse hæmorrhage from the stomach and bowels, in consequence of the rupture of an aneurism of the superior mesenteric artery into the duodenum. Neither was jaundice noted in any of three cases of aneurism of the superior mesenteric artery, recorded in the Pathological Transactions.[1] Two cases, however, of superior mesenteric aneurism are related by Dr. J. A. Wilson in the Medico-Chirurgical Transactions,[2] in one of which large quantities of blood were vomited, while the other 'by pressure on the hepatic apparatus during life had induced jaundice.' A very interesting case also is recorded by Dr. W. T. Gairdner, where jaundice was produced by an aneurism of the superior mesenteric artery, which opened into the duodenum

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quoted from Stokes, as observed by Dr. Beatty, was an aneurism of the aorta, not of the hepatic artery.

[1] Dr. J. W. Ogle's case, vol. viii. p. 168; Mr. Holmes's case, vol. ix. p. 172; and Dr. Wilks's case, vol. xi. p. 44.

[2] Vol. xxiv. p. 221.

22 months before death, and caused repeated and very copious hæmatemesis with symptoms closely resembling those of gastric ulcer. From this case Dr. Gairdner concluded: 'that the combination of jaundice with symptoms indicating imperfect duodenal digestion (cardialgia, pain and vomiting some time after taking food) should in all cases lead to the strong suspicion of a tumour pressing on the ducts of the liver and pancreas near their duodenal termination; that the co-existence of these symptoms with fixed pain or oppression at the epigastrium, pulsation in the same region, and hæmatemesis, would very probably indicate aneurismal tumour, even in the absence of more unequivocal signs.'[1]

8. *Accumulation of Fæces in the Bowels.*

Great accumulations of hardened fæces in the bowels may also compress the bile-duct, so as to cause jaundice and lead to serious errors in diagnosis, hardened scybala being supposed to be nodules of cancer. Dr. Bright has recorded several instances of fæcal accumulation in the colon, mistaken for enlargement of the liver or malignant tumours, and in one of them there was jaundice which disappeared after free evacuation of the bowels.[2] Frerichs also relates a case where an enlargement of the abdomen from fæcal accumulation was at first ascribed to a pregnant uterus, and subsequently, on the supervention of deep jaundice, to an enlarged liver, but where purgatives dispelled the patient's anxiety about a diseased liver and at the same time her hopes of a child.[3] Errors in diagnosis from this cause are all the more likely to arise, inasmuch as great fæcal accumulation may occur notwithstanding that the bowels have acted daily or even been relaxed. They may be avoided, however, by attention to the following rules:—

1. On careful manipulation, the doughy consistence and irregular outline of the fæcal mass will often distinguish it from all other abdominal tumours.

2. In all cases of doubt, the judicious use of laxatives and enemata will get rid both of the tumour and of the jaundice.

9. *A Pregnant Uterus.*

Cases have been frequently observed where the presence of a pregnant uterus, often in conjunction with constipated bowels, has caused jaundice, the course of which will be recognised by its appearing in the advanced stages of pregnancy and disappearing after parturition.

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[1] Clinical Medicine, 1862, p. 504.

[2] Abdominal Tumours. Syd. Soc. Ed. p. 243.

[3] Op. cit. vol. i. p. 69.

10. *Ovarian and Uterine Tumours.*

Tumours of the uterus and ovary have in rare instances been known to compress the bile-duct and cause jaundice. It is sufficient here to mention the fact, as the diagnosis of these diseases from other cases of obstruction of the bile-duct can seldom be difficult.

Prognosis in Jaundice from Obstruction of the Bile-duct.

The prognosis in jaundice from obstruction of the bile-duct will depend mainly on the cause of the obstruction. If the cause be a disease which is of itself mortal, such as cancer, the jaundice will be of secondary moment as regards prognosis. As to the jaundice itself the prognosis will vary according as the obstruction is one that admits, or does not admit, of removal. In both cases it is a matter for enquiry, how long a person can live with obstruction of the common bile-duct. For some months but little inconvenience may be experienced, but usually death from exhaustion takes place within eighteen months, the fatal result being often preceded and hastened by hæmorrhage from the bowels or cerebral symptoms. It is also worth enquiring how long a gall-stone may be impacted in the common bile-duct, and yet ultimately escape, so that the jaundice disappears and the patient recovers. It is remarkable how few observations there are on record, enabling one to give a satisfactory reply to this question. Some years ago I was consulted in the case of a gentleman aged 56, who had deep jaundice from an impacted gall-stone for twenty months, and at the end of that time completely recovered. Dr. Ramskill has recently reported the case of a man who lived for 2½ years with jaundice from an impacted gall-stone;[1] and Dr. Budd has recorded the case of a man who had jaundice for four years from what appeared to be closure of the common bile-duct, and at the end of that time was tolerably stout and muscular;[2]

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[1] Lancet, March 11, 1876. The patient recovered, and for eighteen months was well and free from jaundice (excepting a temporary attack after biliary colic), but died at last of pyæmic hepatitis.

[2] Dis. of Liver, 3rd ed. 1857, p. 233.

but Case CXVIII. seems to show that jaundice from gall-stone, after lasting continuously for nearly six years, may completely disappear. In connection with this subject it is interesting to observe that when the common bile-duct is ligatured in one of the lower animals, the bile after a time finds a passage into the intestine outside the ligature.[1]

Treatment of Jaundice from Obstruction of the Bile-duct.

It has been truly observed that in no ailment have remedies so worthless and absurd been extolled for their efficacy as in jaundice. The patient gets well, and the remedy last tried, such as taraxacum or yelk of eggs, is said to have cured him. All scientific and really successful treatment must be directed against the cause of the jaundice.

The treatment for jaundice arising from obstruction of the bile-duct may be considered under two heads, viz.:—

A. Those measures which are calculated to remove the obstruction; and

B. The remedies which are most likely to alleviate the effects of the obstruction.

A. The measures to be adopted for the removal of the obstruction must depend on its nature. Some of the causes of obstruction are removable, while others are not. It may be well therefore to refer to the several causes of obstruction in succession, and first with regard to—

a. Gall-stones.

Under this head we have first to consider what are the best means for expediting the passage of the stone and preventing its impaction, for the longer time the calculus occupies in its passage, the more likely is it to produce ulceration and stricture of the bile-duct, and become permanently arrested; and secondly, to enquire if there be any remedies which have the power of obviating the formation of fresh concretions, or of dissolving those which already exist in the gall-bladder or biliary passages.

I. *Measures for expediting the passage of the Gall-stone.*

1. When, from the symptoms which I have already described to you, there is reason to believe that a gall-stone is

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[1] Sir B. Brodie, Quarterly Journ. of Science, London, 1823, vol. xiv.; and Dr. Wickham Legg, Barth. Hosp. Rep. vol. ix. 1873.

passing along the bile-duct, it will be well when possible to put the patient in a warm bath, and in all cases to apply heat locally in the form of warm fomentations and poultices.

2. If there be acute tenderness on pressure over the gall-bladder, and the attack has lasted long, great relief will often be obtained from the application of a few leeches over the region of the gall-bladder.

3. Along with these measures you must have recourse to full and repeated doses of opium or morphia. In consequence of the vomiting these remedies ought to be given in the form of pill. A grain of solid opium or quarter of a grain of morphia may be given every two hours until the pain subsides; or, what is still better, from the rapidity of its action, half a grain of morphia may be injected beneath the skin of the arm, and the operation may be repeated from time to time according to its effect. When the patient is at a distance from medical advice, morphia may be administered in the form of suppository.

4. Belladonna is another remedy which often gives great relief, and is especially useful when opium is from any cause contraindicated. Half a grain of the extract may be given every two hours; or a suppository containing half a grain of morphia and one grain of extract of belladonna, or a subcutaneous injection of one sixtieth of a grain of atropine with quarter of a grain of morphia may be used every two hours until the pain ceases. Belladonna and chloroform liniment, applied as a fomentation over the liver under oil-silk, also sometimes affords great relief.

5. Chloroform and ether given by the mouth, or better in the form of inhalation, have also been found to be most efficacious; and they possess this advantage, that while they relieve pain, diminish spasm, and are rapid in their action, as in the case of the uterus in parturition they do not interfere with that muscular contraction which probably assists in the onward propulsion of the stone.

6. Immediate relief is sometimes afforded by large draughts of hot water, containing from one to two drachms of bicarbonate of soda to the pint. According to Dr. Prout, who first recommended this plan of treatment, 'the alkali counteracts the distressing symptoms produced by the acidity of the stomach, while the hot water acts like a fomentation to the seat of pain. The first portions of water are commonly rejected almost immediately; but others may be repeatedly taken, and after some

time it will usually be found that the pain will become less, and the water be retained. Another advantage of this plan of treatment is, that the water abates the severity of the retching, which is usually most severe and dangerous where there is nothing on which the stomach can react. This plan does not supersede the use of opium, which may be given in any way deemed most desirable; and in some instances a few drops of laudanum may be advantageously conjoined with the alkaline solution, after it has been once or twice rejected.'[1]

7. Vomiting of the food which is in the stomach does not require to be checked, but when there is frequent and severe retching attended with pain, its continuance will lower the vital powers and increase the danger of rupture of the distended gall-bladder or bile-ducts, and it must be checked by effervescing draughts, hydrocyanic acid, and ice. On the other hand, when the jaundice is persistent and all symptoms of biliary colic have long ceased, an emetic may dislodge the impacted stone and favour its propulsion.

8. Purgatives are of little use in expelling the stone, and will exhaust the patient; but after the paroxysm of pain is over, saline and mercurial purgatives are usually required to counteract the constipating effect of the opium and to relieve the congestion of the liver.

9. Antimony was long ago recommended by Dr. Bright, with the object of relaxing spasm, but it must be used with caution, as it is apt to increase the sickness and add to the exhaustion which is the patient's main danger.

II. *Measures for dissolving or preventing the formation of Gall-stones.*

1. There are certain remedies which are believed to have the power of preventing the formation of fresh stones, or even of dissolving those already existing in the gall-bladder. A combination of ether (three parts) and turpentine (two parts), proposed by Durande, a physician of Dijon in France, for a long time enjoyed a reputation on the Continent for this purpose; and within the last few years, another French physician, Bouchut, has claimed the same virtue for chloroform administered internally.[2] But although both chloroform and ether will dissolve

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[1] On the Nature and Treatment of Stomach and Urinary Diseases, 3rd ed. 1840, p. 263.

[2] See also Dr. Barclay, Brit. Med. Journ. Jan. 15, 1870.

cholesterin, which is the main constituent of gall-stones, out of the body, neither can reach the gall-bladder or bile-ducts in a sufficiently concentrated form to accomplish this object during life, and the good effects which were thought to follow their use must be ascribed to their antispasmodic properties, and to the relief which they afford to flatulence.

2. It is possible, however, that gall-stones may be dissolved. Concretions are occasionally seen whose surfaces exhibit unmistakable signs of erosion. The remedies which are believed to possess this power in the most marked degree are saline purgatives, alkalies, and diluents. You will do well then to give your patients who have suffered from gall-stones the salts of soda and potash, such as the sulphate, the tartrate, the phosphate, and the bicarbonate, the sulphate of magnesia or the chloride of ammonium, largely diluted, or, what is still better, when practicable, send them to drink the saline and alkaline mineral waters of Carlsbad, Marienbad, Homburg, Vichy, Contrexeville, &c. Although it must be confessed that the evidence of the efficacy of mineral waters and alkalies in dissolving gall-stones is inconclusive and must remain so, there can be little doubt that they improve the general health, lessen the tendency to acid dyspepsia and gout, reduce congestion of the liver, and produce such changes in the bile as lessen the chances of the formation of fresh concretions. In dogs, for instance, with biliary fistulæ, the mere drinking of large quantities of water will increase the amount of water in the bile, and there is evidence that the quantity of soda in the bile may also be increased by taking it into the stomach. From the frequency also with which attacks of biliary colic occur during or immediately after a course of saline or alkaline mineral waters, it would seem that in some way they determine a crisis in the case and favour the expulsion of the stones.

3. In all cases it will be necessary to attend to the patient's digestion and general health. Small doses of blue pill are sometimes very useful. According to Dr. G. Budd, no medicine in some cases does such signal good. 'It seems to increase the quantity of bile and at the same time to render it more healthy, and certainly often improves in a striking manner the general health.'[1] This statement is in complete harmony with my own experience.

4. Lastly, it will be necessary to counteract those habits on

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[1] Op. cit. p. 387.

the part of the patient which experience has shown to conduce to the formation of gall-stones. He must rise early and take plenty of exercise in the open air, sleep in an airy bedroom, live sparely, drink little or no wine, and avoid all rich, fatty, and saccharine food and malt liquors.

b. Hydatids, Distomata, and other foreign bodies in the Bile-ducts.

These causes of obstruction of the bile-duct must be treated on the same principles as gall-stones, with anodynes and antispasmodics. The bursting of a hydatid tumour into the bile-duct is usually preceded by more or less peritonitis and followed by inflammation of the hydatid, which will call for absolute rest, leeches, warm fomentations and opiates. (See Cases XXXII. to XXXIV. p. 112.)

c. Inflammation of the Bile-ducts.

The treatment for obstruction of the bile-duct by inflammatory obstruction of the lining membrane with exudation in the interior has been considered in a former lecture (p. 154).

d. Organic Stricture and Tumours of the Bile-duct.

For the various forms of organic obstruction of the bile-duct arising from stricture or obliteration of the duct or from tumours growing in its interior, no treatment is likely to be of any avail. The obstruction is irremovable, and the jaundice is permanent. In cases, however, where there has been a history of syphilitic peri-hepatitis, mercury and iodide of potassium deserve a trial.

e. Pressure on the Bile-duct from without.

When the obstruction is due to pressure on the duct from without, the treatment must vary according to the compressing cause. Some of these causes are removable; others are not. When the pressure is due to an abscess or hydatid of the liver, or to an ovarian cyst, it will be removed on evacuation of the fluid contents of the tumour; but the pressure of cancerous nodules projecting from the liver, enlarged cancerous glands in the portal fissure, tumours of the stomach, pancreas, kidney, omentum, and uterus, and of abdominal aneurisms, cannot be influenced by treatment. When the symptoms point to waxy or tubercular glands in the fissure of the liver as the cause of pressure, improvement may sometimes be observed to follow the use of iodide of potassium, iron, nitro-muriatic acid, and cod-liver oil, &c. (see Lect. II). Fæcal accumulations in the colon are to be got rid of by castor oil, the administration of frequent small doses of extract of belladonna, and copious oleaginous or warm-water enemata. Lastly, when jaundice shows itself during pregnancy care must be taken to ascertain whether the pressure of the gravid uterus be not aggravated by accumulation of fæces in the bowels.

B. In the next place we have to consider what are the most suitable remedies for relieving the effects of irremovable or persisting obstruction of the bile-duct.

1. One of the first effects of complete obstruction of the bile-duct, if it be not speedily removed, is the accumulation of bile in the bile-ducts and gall-bladder, which become greatly distended and sometimes inflamed in consequence (see *antea*, p. 160), and under these circumstances advantage will often be derived from leeches to the right hypochondrium or round the anus, warm poultices, laxatives, diuretics and diaphoretics, and from taking as little fluid as possible in the way of drink.

2. The diet in all cases requires careful regulation. It ought to be easy of digestion and mainly nitrogenous. Oleaginous and saccharine matters and malt liquors ought to be for the most part excluded.

3. The bowels will require attention. In most cases they are constipated and laxatives will be necessary, and of these the best is a combination of the compound colocynth or rhubarb pill (gr. vi) with blue pill (gr. ij) and extract of henbane (gr. ij). No good can be expected in such cases from remedies which stimulate the action of the liver, or from a course of mercury even supposing that mercury had the power to do this; but there can be no objection to the use of both mercury and podophyllin, as an occasional purgative. Practically their use in moderation is not attended with those injurious consequences which have been theoretically ascribed to them. Although under ordinary circumstances they produce bilious stools, there is no evidence, as I have already pointed out to you, that mercury increases the amount of bile secreted by the liver (p. 330).

4. Flatulence and other dyspeptic symptoms will in many cases call for treatment. The flatulence will often be relieved

by the ethers and essential oils, the gum-resins of assafœtida and galbanum, and by vegetable charcoal; but in most cases the best remedies are those which have antiseptic properties. Bile is an antiseptic, and its withdrawal from the bowels entails decomposition of their contents with generation of gas, but this decomposition will be prevented by the use of such remedies as creasote, turpentine, and carbolic acid (see p. 215). Flatulence and other dyspeptic symptoms arising from the want of bile in the bowels are also often greatly relieved by the use of purified bile from the ox or pig, which may be given in doses of from three to six grains about two hours after meals. As it is not desirable that the bile should come in contact with the stomach, it is well to give it enclosed in capsules, or in pills coated with a solution of tolu in ether. The cholate of soda, of which ten grains may be taken in peppermint water, has also been found useful for the same purpose. The alkalies and mineral acids (p. 137), but more commonly the former, in conjunction with calumba, or with taraxacum, chiretta, gentian, or quinine, are also often of use for improving the appetite and digestion.

5. In all cases of jaundice from obstruction of the ducts it is important to attend to the functions of the kidneys and skin. The kidneys are the principal channels by which the accumulated bile is got rid of from the system, and any disease of these organs (as in Case LXV. p. 159 and Case CV. p. 293) will add greatly to the patient's danger. Persons suffering from jaundice due to obstruction of the bile-duct must avoid sudden chills, and will be benefited by occasional warm baths and by the use of the chloride of ammonium, diaphoretics, and diuretics.

6. The itchiness which is often a source of much distress will sometimes be alleviated by warm baths, the use of a flesh-brush, and the internal administration of bicarbonate of potash (see p. 318). A medical man, who had suffered greatly from itchiness and jaundice due to gall-stones, informed me that among the numerous remedies which he had tried, he had experienced the greatest relief from acetic acid baths (½ pint of acid to 3 gallons of water), or from a lotion of chloroform (1 part) and glycerin (5 parts). Olive oil, calomel ointment, or lotions containing the perchloride or cyanide of mercury (gr. iv to ℥j) or the carbonate of potash or cyanide of potassium (ʒj to Oj), are also sometimes useful. But too often all treatment fails to

give relief, and recourse must be had to opiates or other anodynes to procure sleep.

7. When there is great debility, or when the patient suffers from boils or carbuncles, improvement will sometimes follow the use of the mineral acids with nux vomica or bark, and it will be necessary to allow small quantities of alcoholic stimulants. Of these the best are hock, dry sherry, sound claret, and diluted brandy or gin.

8. When cerebral symptoms supervene, the treatment which has been found most efficacious consists in sinapisms to the nape or scalp, and purgatives. It will be well also to act on the skin by means of diaphoretics, the warm bath, or, what is better, by the hot-air bath, and especially if there be no albumen in the urine to give diuretics.

9. Occasionally, the treatment may have to be modified in accordance with symptoms arising from the disease to which the obstruction of the bile-duct is due, as, for instance, in cases of cancer of the stomach, duodenum, or pancreas, or of abdominal aneurism.

10. Lastly, it is well to remember that in those cases where you succeed in removing the obstruction, the jaundice of the skin and conjunctivæ may persist for a considerable time afterwards, and that then its departure will be expedited by warm baths, diaphoretics, purgatives, and diuretics, and also by benzoic acid, which may be given in doses of four grains made up into two pills with a little glycerin, three times a day.

I shall now proceed to recall to your notice the particulars of a few cases of jaundice from obstruction of the bile-duct, which, for the most part, have been under your observation in the wards.

The first case was a typical illustration of jaundice from gall-stones. The case was also remarkable for the large size of the stone which passed through the bile-ducts into the bowel.

CASE CXVI.—*Biliary Colic—Escape of a very large Gall-stone by the Common Duct.*

Elizabeth G——, 31, adm. into St. Thomas's Hospital April 3, 1872. Five years before had been suddenly seized with violent paroxysmal pain in the right hypochondrium, stretching round to back, accompanied by retching, and followed by deep jaundice, itchiness of skin, and white stools. The pain and retching subsided after five or six hours, but the jaundice persisted for three months. At end of attack biliary concretions were found in stools. After eighteen months she had a similar but less severe attack, the jaundice lasting only one week. Five weeks before admission she had a third seizure, and ever since she had had daily recurring attacks of pain in right side, quite as severe as upon first occasion, attended by retching and sometimes lasting seven hours. At beginning of attack skin had been slightly yellow for a day or two, but after this there had been no jaundice.

On admission, conjunctivæ white, no jaundice nor itchiness of skin, and no bile-pigment in urine. No enlargement of liver nor perceptible bulging of gall-bladder, but marked tenderness corresponding to its fundus. Bowels costive ; motions contain bile.

Patient was ordered an aperient draught of sulphate and carbonate of soda every morning, and for some days felt better. On April 7 and 9 had paroxysms of pain, but not very severe. On April 16 a paroxysm set in much more severe than any before. For more than four days the pain was incessant, and was only relieved by repeated injections of morphia under the skin. Patient, who had borne several children, declared that pain of labour was nothing to this pain. The vomiting also was urgent ; stools contained no bile, and on second day of pain patient became deeply jaundiced. The last paroxysm of pain occurred on night of 20th, and on night of 21st patient passed two facetted gall-stones, about size of small cherries, in a motion containing plenty of bile, and a day or two afterwards a third gall-stone was found in stools, globular and *fully two inches in circumference.* For some days patient had much aching and tenderness in region of liver, but she had no return of severe pain, jaundice speedily subsided, and on May 4 she was able to leave hospital.

Case CXVII. was very instructive as showing how long biliary colic may exist without any jaundice, the explanation in this case being the detention of the stone from its great size in the cystic duct. It was also a rare example of death from sheer exhaustion in uncomplicated gall-stones. The post-mortem examination showed that if the patient could have survived a few days longer, the stone would have passed into the bowel and she would have recovered.

CASE CXVII.—*Fatal Jaundice from Obstruction of Bile-duct by a large Gall-stone.*

In Oct. 1869, a lady about 45 years of age consulted me at my house, and gave following history. For about thirteen years she had been liable, at long intervals, to severe attacks of biliary colic—pain in region of liver, coming on in violent paroxysms, accompanied by vomiting and followed by jaundice, which lasted for a few days. During these attacks her medical attendant had often noticed a painful swelling, corresponding to fundus of gall-bladder.

In autumn of 1868 she had an unusually severe and protracted attack. About Christmas 1868 the attacks of pain became more frequent; they came on almost every day without exception, usually about four o'clock in afternoon, and lasted for twelve hours. The pain was accompanied by vomiting, but all this time there was no jaundice. In April 1869 paroxysms of pain and vomiting became even more frequent and much more severe, and patient was reduced to an alarming state of prostration, from which for many days she was not expected to rally. At this time liver was found to be considerably enlarged and deep jaundice set in, which, though varying in intensity, never disappeared, motions from that date being devoid of bile-pigment. For six weeks during April and May patient was confined to bed, and although for some months before I saw her she had been able to go about, she continued to lose flesh and suffered much from itchiness of skin, flatulence, and almost constant diarrhœa. She was also still liable to attacks of pain and vomiting, though less severe and less regular in their recurrence.

I found her, at time of her visit to me, very thin and weak and deeply jaundiced. Liver was greatly enlarged, but not tender; some tenderness, however, over a rather firm tumour, about size of an orange, corresponding to gall-bladder. No ascites and no enlargement of spleen. The lady had already consulted several medical men of eminence, and more than one had expressed opinion that, whether there were gall-stones or not, there was a cancerous tumour in fissure of liver. This view appeared to me to be negatived by absence of ascites or of any symptom of portal obstruction, while whole history of case seemed to point to a large gall-stone, which from Christmas 1868 to April 1869 had made vain efforts to pass through cystic duct, and thus accounted for attacks of biliary colic without jaundice; but which, during severe attack in April, passed into common duct and produced enlargement of liver and permanent jaundice, with mal-assimilation and emaciation. If this were case, it seemed possible—though, considering duration of jaundice, not very probable—that cause of obstruction might be discharged into bowel and patient get well. I prescribed alkalies and creasote pills to relieve flatulence, and gave instructions that, in event of severe pain coming on, recourse should be had to warm baths, opium, and chloroform.

On afternoon of same day, probably owing to fatigue and shaking consequent on journey from and back to country, pain and vomiting returned with great severity, and continued to recur at short intervals until death from exhaustion three weeks afterwards. During last week of life hæmorrhages occurred from different mucous membranes, and on one occasion, a few days before death, during a violent fit of retching, patient felt a sudden sharp pain in region of gall-bladder, as if something had burst, and soon after she vomited some blood mixed with mucus.

A post-mortem examination was made by Mr. Taylor of Guildford, who kindly furnished me with account of appearances found, and with opportunity of exhibiting to the Pathological Society the obstructed bile-duct. There was nowhere any cancerous deposit. Liver was uniformly enlarged and gorged with bile. But most remarkable appearance was enormous dilatation of cystic and common bile-ducts, which admitted tip of index finger. Duodenal end of common duct was blocked up by a cylindrical gall-stone, measuring about an inch in length and half an inch in thickness. This projected into bowel, where a portion of its surface was bare and exposed from ulceration or rupture of superimposed mucous membrane. Orifice of duct was not enlarged, and could be seen like a little dimple in centre of projection caused by gall-stone. It seemed probable that the mucous membrane stretched over gall-stone had given way, during the attack of sudden sharp pain in region of gall-bladder followed by vomiting of blood and mucus, shortly before death. Seven smaller polyhedral concretions in gall-bladder and dilated cystic duct; two others in hepatic duct, and two in dilated ducts in interior of liver. All had several facets, and had probably been formed in gall-bladder. Blood dark and fluid; extravasations in different parts of body.

Case CXVIII. is an example of recovery after very persistent jaundice from gall-stones. The case was also remarkable for being complicated with extensive xanthelasma.

CASE CXVIII.—*Persistent Jaundice from Gall-stones—Recovery after nearly six years—Xanthelasma.*

Mrs. S——, about 40, from Sydney, consulted me on April 4, 1871. Two years before, when seven months pregnant, began to suffer from severe attacks of pain commencing suddenly at epigastrium, and shooting to back and right shoulder. Pain would last several hours and then cease suddenly, and might return two or three times in a week; it was sometimes attended by vomiting, but there was no jaundice. Two months after confinement had an unusually severe attack of pain, followed by intense jaundice and white stools which lasted three weeks; and for nearly a year these attacks were so frequent that jaundice of one attack had scarcely disappeared before another came on. During the twelve months before I saw her, she had had only one attack of pain, but there had been persistent jaundice, and she had had frequent rigors, which were always followed by an increase of jaundice, darker urine, and whiter stools. She had lost flesh and suffered much from flatulence and tightness and oppression after food, and from attacks of diarrhœa with light fetid stools; and when I saw her she was rather deeply jaundiced, with large patches of xanthelasma on eyelids and neck; urine was as dark as porter, but motions contained no bile; liver protruded about two inches beyond ribs, its lower margin being hard and sharp, and projecting from this was a rounded swelling about size of a small pear, in situation of gall-bladder.

The diarrhœa was always checked by creasote or ox-gall, and under these and other remedies patient improved, gained flesh and strength, and lost dyspeptic symptoms. On July 26 jaundice was very slight and urine contained very little bile-pigment, but there was never any decided sign of bile in stools. After this, patient travelled about and was able to bear considerable fatigue; the jaundice and amount of bile in urine varied from time to time, but there was never any clear evidence of bile in stools. On Dec. 30, after dinner, she suddenly felt sick and faint, and although she had no pain she was for some days after more yellow. On Feb. 2, 1872, after a short walk, she was suddenly seized with most acute pain in region of gall-bladder, shivering, nausea, profuse perspirations, and great restlessness. These symptoms were only relieved by large opiates, and next morning jaundice had increased, and in evening of Feb. 3 there was a recurrence of the pain and other symptoms. For a week after this patient was extremely prostrate and feverish, had frequent retching and deep jaundice and acute pain at tip of right shoulder, with much fulness and tenderness in region of gall-bladder. In night of Feb. 9 she was suffering so much that she had a subcutaneous injection of morphia, which at once relieved her; and next morning she vomited a quantity of green bile for first time for nearly two years. After this she improved; on Feb. 14 she was up and about; jaundice almost, though never quite, disappeared, and motions contained bile. No stone was found in motions, but they were not examined with sufficient care. On May 26 had an attack similar to that on Feb. 2, but less severe, yet followed by fever, tenderness about gall-bladder, and increased jaundice. A few days after this patient returned to Sydney; but I continued to hear of her from time to time. Before she left I ascertained that liver was about an inch smaller than it had been a year before. She was still rather deeply jaundiced.

Until end of 1874 she continued much in same state as when at home, except that she was much weaker. She had no severe pain, but she had frequent fainting attacks followed by an increase of jaundice, from which, however, she was never quite free. But in 1875 jaundice quite disappeared, and general health and strength improved; and in 1876 this improvement continued.

In a former lecture I have called your attention to the difficulty there often is in distinguishing between jaundice from gall-stones and jaundice from cancer (p. 213). In both there may be intense jaundice, no bile in the stools, great emaciation and weakness, and paroxysms of severe pain. The difficulty is increased by the circumstance that cancer of the liver is a common sequel of gall-stones. At Guy's Hospital, indeed, it has been found that when death occurs in persons with gall-stones, it is most frequently brought about by the development of cancer about the gall-bladder or bile-ducts.[1] Case CXIX. was further interesting from the circumstance that although the obstruction of the common bile-duct was not removed, bile appeared in the stools before death owing to the establishment of a fistulous passage between a dilated bile-duct and the stomach.

CASE CXIX.—*Gall-stone impacted in common Bile-duct.—Fistulous opening of dilated Bile-ducts into Stomach—Cancer of Liver.*

Elizabeth W——, aged 64, adm. into St. Thomas's Hosp. Jan. 21, 1876. No history of cancer in family could be made out. A sister had died at 54 of asthma. Patient had been of temperate habits, and had enjoyed good health until present illness. For four years she had suffered from attacks of severe pain in region of gall-bladder, recurring at intervals of four, five, or six months. The pain would come on in paroxysms for three or four days and was severe, so that she was laid up for ten or fourteen days, but it was never attended by vomiting nor followed by jaundice. Towards end of July 1875 she had an unusually severe and protracted attack, and after six weeks from commencement of attack, although pain had subsided, jaundice appeared for first time and persisted, and from this date she lost appetite, flesh, and strength. She still suffered from indefinite pains, but after appearance of jaundice she never had the severe paroxysms such as she had had before. Five weeks before admission, she was attacked with diarrhœa (light coloured stools), which ceased after three weeks. For four weeks she had had a frequent harassing cough.

On admission, emaciated and deeply jaundiced. Tongue slightly coated, not too red; appetite improved; bowels regular; motions reported as light-coloured. In right nipple line liver did not seem to extend beyond margin of ribs, but between this and left nipple line could be distinctly felt a solid mass, apparently continuous with liver, and extending to 2½ in. below umbilicus, smooth, firm, and not tender, although pressure upon it caused pain in back. Lower part of chest contracted from lacing. No ascites. Urine 1015, loaded with bile-pigment, but no albumen. Pulse 84, weak; heart-sounds healthy. Frequent cough with muco-purulent expectoration; a few sibilant râles over front of both lungs; posteriorly over lower fourth of left lung, marked dulness, distinct tubular breathing, diminished vocal resonance, and coarse moist râles. Temp. 96·8° to 99·2°. Frequent headache.

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[1] Fagge, Guy's Hosp. Rep. 1875, vol. xx.

Patient was treated with nux vomica and mineral acids, and a nutritious diet with a moderate amount of stimulants. At first she improved a little; but on Jan. 31, weaker; much pain in back; appetite less; bowels costive; distinct evidence of bile in stools; jaundice unchanged; sleeps badly. Under draughts of morphia at night slept better and again improved a little, and on Feb. 26 dulness was noted as having quite disappeared from base of left lung, where there was now vesicular breathing. But after this became rapidly weaker, and pain in back was more severe. On March 9 she vomited a quantity of dark liquid, passed into a state of collapse, and died same evening. All time she was under observation pain never had characters of biliary colic.

Autopsy.—Body very emaciated. Peritoneum contained a small quantity of fluid; abdominal organs adherent by a thin layer of recent lymph. Liver much displaced, left lobe occupying greater part of right half of abdomen, reaching 6 in. below ribs in right nipple line; right lobe displaced upwards and backwards, pushing up right half of diaphragm, and not visible from front of abdomen. Left lobe thin and contained several small nodules of cancerous growth. Gall-bladder moderately distended with colourless, nearly clear mucus. Common bile-duct contained similar mucus, and uniformly distended to about five lines in diameter; about 1 in. from its termination in duodenum a calculus, size of a small marble, was found impacted, and above this were several smaller dark concretions. A probe passed upwards into hepatic duct entered a large mass of cancerous growth, occupying centre of liver and extending forwards to neighbourhood of gall-bladder, hard and fibrous externally, but in centre much degenerated and containing calcareous masses. Bile-ducts throughout liver, but especially those in left lobe, greatly dilated and filled with thick bile, some of dilated ducts projecting from surface of liver. Stomach contained a quantity of greenish-yellow mucus giving reaction of bile-pigment. Anterior wall of stomach adherent to under surface of left lobe of liver and at adherent portion were three or four small rounded orifices, from one to two lines in diameter, by which stomach communicated with dilated bile-ducts in superficial portions of liver. On squeezing liver, bile flowed through them into stomach. This was source of bile found in bowels and in stools during life. Spleen somewhat enlarged. Signs of recent inflammation with a pint of turbid fluid in right pleura. Numerous small nodules of new growth on pleural surface of right half of diaphragm and in substance of right lung. A small quantity of lymph on lower lobe of left lung.

In Case CXX. there was also a concurrence of gall-stones with cancer of the liver and pancreas.

CASE CXX.—*Conjunction of Gall-stones and Cancer of Liver and Pancreas—Jaundice.*

Sarah H——, 53, adm. into St. Thomas's Hosp. Nov. 4, 1872. Father died at 70, and mother at 76; causes of death unknown. Four brothers and four sisters alive and well; none dead. Patient had enjoyed good health until two months before admission, when she was suddenly seized with violent pain at epigastrium and between shoulders, attended by shivering and vomiting. Pain was paroxysmal and lasted one week, and then suddenly ceased. After about a fortnight she had a second attack of pain, vomiting, and rigors, lasting for a day or two, but subsequently to this there was no recurrence of these symptoms. On second day of first attack she was observed to be yellow; and from then up to admission jaundice had increased in intensity.

On admission, no pain and not very emaciated; deep jaundice; no itchiness; tongue slightly coated; appetite good; great flatulence, but no vomiting; four or five loose, offensive, clay-coloured motions in day. Distinct fulness and tenderness in region of gall-bladder; this could be felt to project beyond anterior margin of liver, which was not much enlarged. Urine loaded with bile-pigment. Pulse 80, irregular and intermittent; heart healthy.

Was ordered meat diet, an alkaline mixture, and a creasote pill twice daily after meals. A few days after admission patient began to complain much of pain across back. On Nov. 13 there was slight ascites and considerable œdema of both legs, the right leg being larger than left. On Nov. 21 the ascites had increased; there was induration of integuments around navel, and pain was so severe that it was necessary to have recourse to subcutaneous injections of morphia. After this, patient rapidly sank, and died on Nov. 26.

Autopsy.—Peritoneum contained two gallons of clear serum. Liver 94 oz., yet it projected but little beyond ribs. Gall-bladder greatly enlarged and forming a prominent tumour. Liver adherent to diaphragm, stomach, duodenum, and pancreas. Projecting from its surface were numerous nodules of cancer up to 1½ in. in diameter, very vascular, and larger ones cupped. Immediately above gall-bladder was a mass of cancer, 3 in. in diameter, infiltrating tissue of liver and involving its whole thickness. Gall-bladder full of pus, with more than twenty gall-stones, but no bile. Cystic duct almost obliterated. Common duct, hepatic duct, and ducts inside liver all much dilated. Growing from head of pancreas was a large tumour surrounding portal vein and encroaching on duodenum where bile-duct enters. Omentum much thickened from cancerous deposit. About a pint of serum in each pleura. Both lungs studded with cancerous nodules from size of a pin's head to that of a pea, the larger ones cupped. Other organs healthy.

In former lectures I have brought under your notice several other cases of jaundice from gall-stones. In Case LXV. (p. 159) the jaundice resulted from inflammation of the biliary passages excited by gall-stones in the gall-bladder, but there had been no history of biliary colic; in Case LXVI. (p. 162) there was jaundice, with enlargement of the liver and gall-bladder, from obstruction of the common duct by a calculus; and in Case LXXI. (p. 173) attacks of biliary colic and jaundice were followed by pyæmic abscesses in the liver and death.

You will remember also that, when on the subject of enlargements of the liver from hydatid tumour, I related to you several cases where the bile-duct became obstructed by hydatid vesicles which had escaped from the ruptured parent hydatid, and that in one of these cases (Case XXXIV., p. 116), the passage of hydatid vesicles along the bile-duct produced all the phenomena resulting from the passage of gall-stones.

I have also in a former lecture called your attention to several cases where jaundice resulted from inflammation of the bile-ducts interfering with the flow of bile (see p. 155).

The next case is an example of a very rare form of jaundice, where the cause was a congenital closure of the bile-duct.

CASE CXXI.—*Jaundice from congenital Closure of Bile-duct.*

Esther W——, aged 2 months, was brought to me by her mother as an out-patient at this (Middlesex) hospital on January 7, 1862. The mother stated that the child had appeared healthy when born, but that a few days afterwards she was noticed to be unusually yellow, and that this yellowness had been increasing, while the child had suffered from diarrhœa and had been emaciating. When first seen by me child was very thin, and skin and conjunctivæ were of an orange-yellow colour. Motions perfectly white and very offensive. A teaspoonful of chalk mixture, to be taken two or three times a day, was all that was prescribed.

On Jan. 21 child not quite so yellow, but thinner, and motions red like brick-dust, evidently from presence of blood. She had also had several attacks of slight epistaxis.

During next week child continued much in same state, still losing a little blood occasionally from nose, and getting gradually thinner, although there was less diarrhœa.

On March 11 it was noticed that several black lumps had appeared over chest and back. They varied in size up to three quarters of an inch in diameter and were considerably raised above surface; they were evidently due to extravasations of blood beneath skin.

On March 25, mother stated that child had been much worse for two previous days, vomiting everything she swallowed, and vomited matters containing blood. The ecchymoses on surface had also increased both in number and size.

Two days after this child died, and on careful dissection common bile-duct was found to be completely obliterated, its place being occupied by a small quantity of areolar tissue. Gall-bladder extremely small and collapsed, and contained only a few drops of colourless fluid. The opening of duct in duodenum was found with difficulty, and a probe could not be passed through it into bile-duct. Liver jaundiced with a few fibrous bands of adhesion on under surface; ducts dilated; in other respects appeared normal. Altered blood in contents of bowels, and several small extravasations beneath mucous membrane of both stomach and intestines.

In a future lecture I shall bring under your notice a case where jaundice was the result of constriction of the duct from peri-hepatitis (Lect. XII.), and Case CXXII., of which I show you here the specimen, dissected by me many years ago while pathologist to the Middlesex Hospital, is a rare example of obstruction of the orifice of the bile-duct in consequence of its being involved in the cicatrix of a duodenal ulcer. In the case from which this preparation was obtained there was painful enlargement of the gall-bladder and jaundice, arising from obstruction of the duct caused apparently by the cicatrisation of a duodenal ulcer. The contraction of the liver in this case was no doubt due to the long duration of the obstruction, the hepatic tissue having become atrophied from the pressure upon it of the permanently distended bile-ducts.

Case CXXII.—*Jaundice from Obstruction of Common Bile-duct by Cicatrix of a Duodenal Ulcer—Dilatation of Bile-ducts and Atrophy of Liver.*

James B——, aged 69, a coachman, of small frame and spare habit, admitted into Middlesex Hospital on May 4, 1861. During greater part of his life he had been in habit of drinking a good deal, but for last seven years he had been a very steady, sober man. With exception of an attack of acute bronchitis nine years before, which left him 'asthmatic,' he had enjoyed good health until four months before admission, when he had been suddenly seized with acute pain in right hypochondrium, vomiting of bitter matter, and much fever. After a fortnight skin became jaundiced and he had great formication. Jaundice increased in intensity, but formication diminished. Latterly he had suffered from palpitation in cardiac region and throbbing in head, and he had lost both flesh and strength. At commencement of his illness he had been for a fortnight in another hospital, where he had been salivated.

On admission, patient was emaciated and feeble; pulse 72, and intermitting; whole skin and conjunctivæ of a bright lemon-yellow. Patient complained of a dull pain in region of liver, dulness on percussion of which appeared considerably increased, measuring upwards of 5 in. in right mammary line. On more careful examination, it was ascertained that this enlargement was limited to situation of gall-bladder, and that posteriorly and laterally hepatic dulness was diminished. Tongue had a yellowish coat. He had no bitter taste, but sense of taste was in a great measure abrogated; no appetite; bowels confined, motions being nearly white. Urine dark, like porter, and contained much bile-pigment. Bladder had to be evacuated by catheter. Over both lungs sonorous rhonchi could be heard, and expiration was prolonged. He still complained of itching of skin at night.

Patient was treated with alkalies, ammonia, vegetable bitters, and stimulants; but prostration rapidly increased, a bed-sore appeared over sacrum; tongue became dry and brown, and sordes collected on teeth; motions became very dark from presence of blood; pulse rose to 90; signs of bronchitis increased, and for several days before death, which occurred on May 19, there was low muttering delirium.

At autopsy, liver small, pale, and flabby; lower margin did not reach so far as edge of ribs. Gall-bladder about four times normal size, and filled with a colourless flaky fluid destitute of any tint of bile. Cystic, hepatic, and common ducts all enormously dilated, common duct being larger than one's finger, and all filled with a colourless fluid similar to that in gall-bladder. Bile-ducts also in interior of liver greatly dilated as far even as outer surface, and filled with a similar fluid, which flowed out when liver was cut into. No calculus in gall-bladder nor in any of ducts, but orifice of common duct in duodenum completely blocked up. Coats of bowel at this part dense and considerably thickened, forming a nipple-like prominence about size of a hazel-nut, and around this mucous membrane had a radiated, puckered appearance, as if from cicatrisation of an ulcer. No ulcers nor cicatrices elsewhere in bowels, and no evidence of morbid deposit in head of pancreas nor in adjacent glands, but pancreatic ducts much dilated. Secreting tissue of liver of an olive-yellow colour and flabby, but not friable; outlines of lobules obliterated, and on microscopic examination much granular and oily matter was discovered, but secreting cells had mostly disappeared. Much atheroma of aorta and of valves of heart. Lungs presented characters of bronchitis and emphysema of old standing. Prostate enlarged and bladder contracted, its mucous membrane being very hyperæmic, encrusted with flakes of diphtheritic exudation as large as a shilling; kidneys granular, and pelves and calices dilated.

When lecturing on the subject of enlargements of the liver from cancer, I brought under your notice several examples of this disease in which there was jaundice. In one (Case LXXXVII., p. 220) there was unfortunately no post-mortem examination; in another (Case LXXXV. p. 216) the jaundice was due to compression of the common duct by a dense mass of areolar tissue and enlarged cancerous glands in the portal fissure.

In Case CXXIII. the jaundice was due to compression of the common bile-duct by a mass of cancerous glands in the fissure of the liver secondary to cancer of the stomach. It was notable that at first the patient gained weight under treatment.

Case CXXIII.—*Cancer of Stomach and Liver, Jaundice, and Ascites.*

Charles D——, labourer, aged 58, adm. into Middlesex Hosp. Sept. 10, 1869. No family history of cancer. For some years had been liable to attacks of diarrhœa from slight causes, but with this exception had enjoyed good health until four months before admission, when he began to suffer from pain at epigastrium and flatulent distension after meals, and he had occasional retching in the morning, but never brought up his food. These symptoms increased in severity, and soon patient lost all appetite for food and became rapidly emaciated. After a month jaundice appeared, and was at first attended with intolerable itchiness of skin. Quite recently his legs had become swollen after walking.

On admission patient was very thin; weighed only 89 lbs. (weight had never exceeded 112 lbs.). Deep jaundice of skin and conjunctivæ, and moderate œdema of both legs. Liver enlarged, in r. m. l. measuring 5½ in. and extending 1½ in. beyond ribs; its surface in epigastrium somewhat tender, with a tolerably distinct projecting nodule, just above umbilicus, also a distinct projection from edge of liver corresponding to gall-bladder. Abdominal veins dilated; slight ascites; splenic dulness not increased; girth at umbilicus 27½ in. Tongue white; immediately after eating has much pain at epigastrium, lasting for half an hour; no vomiting; bowels regular; white stools. Urine loaded with bile-pigment and lithates; no albumen. Sleeps well.

Under use of nitro-muriatic acid and bitter tonics, patient's appetite somewhat improved, and in last fortnight of September he gained 3lbs. in weight. Pain at epigastrium, however, increased; on Sept. 27 another tender nodule could be felt just below ensiform cartilage, and on Oct. 4 a third was appreciable a little to right of second; bowels became loose, ascites and œdema of legs increased, and jaundice persisted. On Oct. 15 girth at umbilicus was 31 in., and patient being much weaker and thinner left hospital for his own home at Castle Hedingham, where he died on Nov. 8.

A post-mortem examination was made by Mr. E. Andrews, to whom I am indebted for following particulars. Three quarts of fluid in peritoneum. Stomach contracted, its walls infiltrated with much cancerous matter deposited between mucous and muscular coats; orifices not involved, and mucous membrane not ulcerated. Left lobe of liver adherent to diaphragm, and scattered through it several cancerous nodules, some of which projected from surface. In fissure of liver was a large mass of hard cancerous matter, compressing portal vein and blocking up cystic and common bile-duct. Thoracic organs healthy.

In the three cases which follow, the jaundice was due to a cancerous tumour which seemed to originate in the head of the pancreas; but in Case CXXIV., as you will see by this preparation (Middlesex Hospital Mus. Cat., Series IX. No. 18), the obstruction of the bile-duct was due to an independent cancerous growth springing from its lining membrane; and in the two others (Cases CXXV. and CXXVI.) it was produced by a mass of cancerous matter in the portal fissure.

CASE CXXIV.—*Jaundice from Obstruction of Common Bile-duct by a cancerous Growth from its lining Membrane—Dilatation of Bile-ducts and enlargement of Gall-bladder—Cancerous Growth in Pancreas.*

The patient from whose body this preparation was obtained was a cabinet-maker, aged 36, who was admitted into this (Middlesex) hospital on Sept. 1, 1857. His illness had commenced on June 1 with vomiting and purging, followed after three or four weeks by intense jaundice, dark urine, and white stools. He had no symptoms of gall-stones, but there was a settled dull pain in region of liver, which was enlarged and tender. He complained of loss of appetite, thirst, and lassitude. He had been treated at first with mercurials, and subsequently with nitro-muriatic acid and taraxacum and saline aperients; and by middle of August liver seemed reduced to nearly its normal size and was free from tenderness, but gall-bladder was enlarged and seemed to be about size of a small pear, and jaundice was increased.

When admitted into hospital, patient had intense jaundice, and urine was loaded with bile-pigment, of which fæces contained not a trace. Liver was of natural size, but gall-bladder could be felt reaching as low as umbilicus and was seat of considerable tenderness. There was much tympanites, but no ascites nor enlargement of abdominal veins. Patient was very emaciated and low-spirited, had no appetite, and passed sleepless nights.

Iodide of potassium, bicarbonate of potash, mineral acids, taraxacum, gentian, quinine, and nitro-hydrochloric acid baths and lotions were tried in turn, but nothing made any impression on the disease. The tumour corresponding to gall-bladder increased in size and became more tender, and about three weeks after admission patient became suddenly unconscious and had symptoms of rapid dissolution, with muttering delirium. These symptoms gradually subsided in course of an hour and did not recur, but patient gradually sank and died on Oct. 21.

Autopsy.—Gall-bladder projected 3 in. beyond anterior margin of liver, and extended to within an inch of anterior superior spinous process of ilium; its length was altogether 7½ in. Its coats were attenuated, and its inner surface had lost its reticular appearance and was rough, red, and granular. It contained 15 fluid oz. of a pale turbid fluid, like barley-water, which on standing deposited numerous small particles of inspissated bile. The fluid was alkaline; its specific gravity was 1010; and it contained numerous very large cells presenting from two to four nuclei with nucleoli, besides flakes of tesselated epithelium, granular matter, &c. Cystic and greater part of common bile-duct also much dilated, latter being large enough to admit a man's thumb, and on cutting into liver a large quantity of white fluid like that in gall-bladder escaped from greatly dilated biliary ducts. Liver weighed 60 oz.; it was of a dark olive-green colour; outline of lobules distinct. On tracing common bile-duct to bowel, it may be seen in the specimen to be obstructed by an exuberant growth of medullary cancer growing from its inner surface, and extending for about 2 in. upwards from duodenum. Before duct was slit up, a probe could be passed with difficulty from duodenal orifice through obstruction. On cutting into growth, a glairy juice exuded, which contained large compound nucleated cells, similar to those found in fluid in gall-bladder, and free nuclei. Head of pancreas was involved in a growth larger than an orange, which on section presented a medullary appearance and yielded a copious thick, milky juice, containing many large nuclei, but no mother-cells. This tumour had compressed bile-duct, but did not encroach upon its walls, and growth in duct appeared to have arisen independently. Heart weighed only 6 oz. All organs and tissues of body deeply jaundiced, but, with exceptions mentioned, in other respects healthy. Contents of bowels devoid of bile-pigment.

CASE CXXV.—*Cancer of Pancreas and of Gall-bladder—Jaundice from Obstruction of Bile-duct.*

At end of May 1866, I was consulted in reference to case of Mr. D——, a gentleman between 60 and 70 years of age, who had jaundice. His illness had commenced about beginning of January, with symptoms of bronchitis, loss of appetite, and restless nights. On one of last days in February he had been exposed to severe cold, and next morning he awoke with intense nausea and retching, which continued till night. Slight febrile symptoms followed; pulse rose ten beats above its average; appetite was capricious, and bowels required aperients. He rapidly lost flesh, and about middle of April jaundice appeared. His symptoms at time I was consulted were as follows:—Deep jaundice of skin and conjunctivæ. Urine scanty and very dark, and contained a large quantity of bile-pigment. Slight tendency to diarrhœa; motions devoid of bile, of a leaden hue and very offensive, but sometimes contained a small quantity of blood from piles. Liver appeared to be quite within its normal limits, but there was occasional uneasiness in right hypochondrium, and decided tenderness with slight hardness about situation of gall-bladder; superficial veins of abdomen slightly enlarged. Pulse 72; increasing emaciation and debility, with decreasing appetite and occasional retching; countenance exhibited an anxious, cachectic aspect.

The treatment consisted in effervescing draughts with hydrocyanic acid and Battley's liquor opii, and in rubbing in externally iodide of potassium ointment.

About beginning of June vomiting became more urgent; almost everything that was swallowed was rejected; pain increased; tongue became thickly furred and brown; and emaciation increased daily. After three days of unconsciousness and low delirium, Mr. D—— died on June 19.

A post-mortem examination was made by Mr. Moreton of Tarvin, Cheshire, to whom I am mainly indebted for following particulars, and for forwarding to me some of parts for examination. Firm bands of adhesion connected whole surface of liver to surrounding parts. No ascites. Gall-bladder about size of a hen's egg, its walls greatly thickened from cancerous deposit, and its cavity containing about a teaspoonful of thick fluid like cream mixed with blood. Opening of gall-bladder into cystic duct closed; and both cystic and common bile-duct were imbedded in, and obstructed by, a mass of cheesy, cancerous matter. No cancerous deposits in liver, but in head of pancreas was a mass of cancer as large as a small orange. All of these deposits gave on section a creamy juice, containing usual cellular elements of cancer.

CASE CXXVI.—*Jaundice from Obstruction of Bile-duct by Cancer originating in head of Pancreas.*

Mary C——, aged 64, adm. into St. Thomas's Hosp. Nov. 2, 1875. Mother had died at 93; father 52, and two sisters, 18, and 36, had died of consumption; two brothers alive, 70, and 65. No family history of cancer, and habits had been temperate. For several years had suffered from aching pains in back and been easily fatigued, and during same period she had suffered from nausea and sometimes from vomiting, usually before breakfast, and from occasional attacks of pain between umbilicus and right ribs. For ten months she had been losing flesh, pain and sickness had been more frequent, and bowels had been constipated. Ten weeks before admission she had an attack of very severe pain in upper part of abdomen lasting 24 hours, and shortly after this she became jaundiced and skin was very itchy. After appearance of jaundice she became more rapidly emaciated, and two weeks before admission legs began to swell.

On admission, weak, emaciated, and deeply jaundiced. Urine loaded with lithates and bile-pigment. Stools light and contain no bile, but stated to be sometimes very dark. Tongue red, smooth, and dry; appetite bad; food gives her pain, and on three occasions since jaundice has vomited; bowels costive. Liver much enlarged in every direction, measuring vertically in right nipple line 8 in., of which 3 below ribs; surface hard, nodulated, and slightly tender; has at times great pain in liver, which may last several hours. Slight ascites and some œdema of legs, more of left than of right.

Was ordered small doses of nux vomica, with occasional anodynes and a light diet. For two or three days bowels were relaxed and motions light-coloured. On Nov. 9 breathing became embarrassed and there was some cough. These symptoms rapidly got worse; lips livid, and all signs of consolidation at lower and back part, first of right, and then of left lung. On Nov. 16, died asphyxiated.

Autopsy.—Cancer of head of pancreas and of glands in hilus of liver, growing into duodenum and involving common and cystic duct. Secondary growths in liver. Extensive hypostatic consolidation of lower and back part of both lungs, with several patches of lobular pneumonia in lower lobe of right, but no sign of new growth. Kidneys somewhat granular.

In the three following cases jaundice was due to cancer taking its origin in the duodenum. The patient whose case I will first relate to you was under your observation for eight months, and you will remember how frequently I called your attention to the distressing itchiness from which he suffered. He was also the subject of boils and carbuncles and of yellow vision (p. 321); but the main source of interest in his case was the cause of the obstruction, about which we often speculated. The improvement which at one time took place in his appearance and weight seemed to negative cancer of the pancreas or duodenum, or indeed cancer of any other organ. Moreover the duodenal tumour, you will observe, occupied exactly the position of the gall-bladder, so that, although there was no clear history of biliary colic, the most probable causes of obstruction, on the man's first admission, appeared to be an impacted gall-stone, a simple stricture of the duct, or closure of its orifice by the cicatrix of a duodenal ulcer. Even at a later stage, the rigors, pyrexia, and night-sweats left it doubtful whether the rapid enlargement and nodulated condition of the liver and the severe paroxysms of pain might not result from pyæmic abscesses of the liver, secondary to ulceration of the bile-duct from the pressure of a large gall-stone; and this view received confirmation from the circumstance, that the advent of these serious symptoms was accompanied by a disappearance of the jaundice and itchiness after a duration of seven months.

CASE CXXVII.—*Cancer of Duodenum—Jaundice from Closure of Bile-duct—Sloughing of Tumour and Disappearance of Jaundice—Secondary Cancer and Abscesses of Liver.*

William M——, aged 50, a porter, but formerly a fireman, admitted into Middlesex Hosp. under my care on Nov. 26, 1867. Twelve years before admission, he had suffered from bronchitis, and four years before he had been laid up for seven weeks with rheumatic fever. For ten years he had been subject to piles, and had occasionally lost a good deal of blood from them. He had been in habit of drinking a good deal of beer, but he had never been a spirit-drinker. There was no history of cancer in his family; his father was alive, aged 83, his mother had died at 63 from rupture of a blood-vessel. Six weeks before admission, he noticed his urine to be very dark, and his motions pale, and that he was becoming thinner and weaker, and about same time he began to suffer from itchiness of skin. After about a fortnight he found his skin becoming yellow, and he often awoke in night with severe pain at pit of stomach, alleviated by friction, which seemed to bring up a quantity of gas. Skin and urine gradually became darker, and on one occasion, a fortnight before admission, he vomited about a pint of clear watery, tasteless fluid.

On admission, patient was thin, and had very deep jaundice of skin and conjunctivæ, but chief complaint was of intense itchiness over whole body, though most severe in palms of hands and soles of feet, which kept him awake at night. Skin was marked by numerous scratches, but no eruption. Urine very dark like porter, and yielded reaction of bile-pigment in a marked degree, but contained no albumen. No feeling of pain or tenderness in region of liver, which did not project beyond edge of ribs, and which did not seem to be materially altered in size, dulness in right mammary line being $3\frac{1}{4}$ in. and man being of small build and short stature. Corresponding to gall-bladder, however, hepatic dulness seemed to project about half an inch from general boundary line. Splenic dulness not increased. No ascites, no appreciable abdominal tumour, no enlargement of abdominal veins, and but slight tympanitic distension of bowels. Tongue moist and coated white. A bitter taste, especially in morning; nausea and loss of appetite; bowels regular; motions clay-coloured. Cardiac and respiratory symptoms normal, except that pulse was only 52. Temperature 97·5°. Weight on Oct. 2, before he began to be ill, had been 132 lbs.; a few days after admission it was 116 lbs.

He was ordered a mixture with bicarbonate of soda (gr. x) and spirit of chloroform (♏xx) three times a day, and a henbane draught at night.

A few days after admission it was ascertained that patient had yellow vision; everything white appeared to him yellow. Conjunctivæ were at same time considerably injected. Yellow vision disappeared about end of December and did not recur, although no change took place either in jaundice or in amount of blood in conjunctival vessels.

Henbane, Indian hemp, opium, subcutaneous injections of morphia, ox-bile, benzoic acid, and warm baths were resorted to in succession, but failed to relieve itchiness. Patient suffered also much from attacks of flatulent colic, which appeared to be relieved by a pill with creasote (♏j) and galbanum (pil. galb. co. gr. iij), taken twice a day, and subsequently by the confectio terebinthinæ. Pills of ox-bile were tried without any result. On Dec. 13, and again on Dec. 28, he vomited breakfast.

On Dec. 30, following note was entered in case-book. 'For first time there is felt what appears to be a deep-seated, hard tumour, about size of a walnut, 1½ in. above and to right of umbilicus, which is not at all tender;' and on Feb. 10 there is this additional note: 'Tumour formerly noted is more distinct; it appears to be about size of a small orange, and situated to right of umbilicus. It is hard, and its surface is slightly lobulated but not at all tender. It is distinctly movable and appears to be continuous above with liver, with which it is connected in precisely situation of gall-bladder. Its lower extremity is fully 2¼ in. below general outline of liver.'

The itching continued to be very intense and was source of great distress; but on three different occasions, viz. on Jan. 20, Feb. 8, and March 18, it was at once, and for many days, relieved by a mixture containing 10 gr. of the bicarbonate of potash with 10 gr. of the nitrate of potash, taken three times a day. During last fortnight of February patient had many attacks of severe abdominal pain, often lasting for several hours and relieved by eructations of gas and fluid. On Feb. 26 he vomited food. Abdomen about this time became moderately distended from tympanites. From first his bowels had been open two, three, or four times a day, motions being of fair consistence, but clay-coloured and very offensive. Jaundice and colour of urine varied in intensity from time to time, latter being sometimes almost black from amount of bile-pigment. Appetite failed entirely, but was considerably improved by quinine pills ordered on Feb. 12. He continued to lose weight until March 4, when he weighed only 105 lbs., being a loss of 11 lbs. since admission; after this his appearance improved somewhat, and on April 1 he had gained 3 lbs. About end of January he suffered for about a week from small, but very painful, boils in meatus of left ear. At beginning of March a crop of small painful boils appeared on back part of scalp, and at end of March a large carbuncle formed over occiput and upper part of neck, on account of which he was transferred for three weeks to one of the surgical wards.

May 1, 1868.—Condition now is as follows. Appearance is on whole improved and he has gained 5½ lbs. in weight since March 4. Jaundice decidedly less and urine contains less bile-pigment, but motions still clay-coloured without a trace of bile. Itchiness has been more distressing than ever since taking a mixture containing nitric acid and bark. Tumour has undergone little change since Feb. 10; it is still hard and painless. Area of hepatic dulness is decidedly less than on admission. Less tympanites and no ascites. Appetite better; bowels still open two or three times a day.

May 27.—Has gained 7 lbs. in weight since March 4. Still suffers much from itchiness, but jaundice is less and urine paler than since admission. For a week has taken three times a day, two hours after meals, a gelatine capsule containing three grains of ox-gall, but motions still clay-coloured and scarcely darker than they were before.

June 6.—Two days ago had a very severe attack of abdominal pain, lasting for two hours, and accompanied by vomiting, perspiration, coldness of surface, and weak pulse. General condition remains the same, and no change in colour of motions.

After this patient again began to lose flesh (June 9, weight 108 lbs.); he became much weaker and suffered from epigastric pain and a burning pain in hands. On June 14 he began to vomit food, and next day motions were black, as from presence of blood. On June 19 he was scarcely able to walk, and tumour was noted as larger and somewhat more to right. On June 25 he vomited frothy matter containing much sarcina, and urine contained a small quantity of albumen and numerous crystals of oxalate of lime. On June 30 he again vomited much sarcina; liver appeared to be larger and its surface in epigastrium was distinctly nodulated. Vomiting of sarcina and melæna continued; prostration and emaciation rapidly increased, but jaundice was much less and only very little bile in urine; and on July 17 lower edge of liver reached to within 1½ in. of umbilicus, he had had a distinct rigor and there was now much pyrexia, pulse having risen from 60 to 96, and temperature being 104·75°; perspirations during sleep; some œdema of feet. On July 19 distinct bulging at epigastrium; lower edge of liver reached quite to umbilicus, and hepatic dulness in r. m. l. measured 8 in. On July 23 weight only 92½ lbs. Pyrexia and perspirations continued; and on July 26 diarrhœa with distinctly bilious motions; there had been no itchiness for three weeks. On July 29 another severe rigor with intense epigastric pain and cramps in limbs. Pain at epigastrium recurred frequently, and patient slowly sank until death on Aug. 3. During last week of life jaundice scarcely appreciable and urine pale and clear.

Autopsy.—Liver greatly enlarged, reaching down to umbilicus, and studded with numerous masses of soft cancer up to size of a walnut; some flakes of recent lymph on its under surface. Below right lobe was a rounded tumour of size of an orange; this occupied situation of gall-bladder, but was unconnected with it, and originated in coats of duodenum. Corresponding to this tumour, on mucous surface of duodenum, was a cancerous ulcer, commencing 2 in. beyond pylorus and extending downwards for 2½ in. Substance of tumour was made up of medullary cancer. Common bile-duct large enough to admit finger, but yet water could be injected from it into duodenum through a ragged sloughy opening in ulcer. Gall-bladder contained an ounce of thin bilious fluid, but no calculus. Bile-ducts in interior of liver much dilated, and many of them seemed to terminate in small abscesses containing thick yellow pus. Pancreatic ducts also dilated. Kidneys congested. All other organs healthy.

CASE CXXVIII.—*Cancer of Duodenum and Stomach—Secondary Deposits in Liver etc.—Jaundice and Ascites.*

Frederick R——, a painter, aged 38, adm. into Middlesex Hosp. Feb. 14, 1870. No family history of cancer. Until nearly three years before admission, when he had an attack of delirium tremens, had been intemperate. At beginning of 1868 had an attack of sorethroat without eruption, but followed by desquamation of skin. On recovering from this, first began to suffer from pain in epigastrium and occasional vomiting, coming on usually two or three hours after food, and these symptoms had persisted ever since with increasing severity. In Aug. 1869 first noticed a hard and somewhat tender swelling in upper part of abdomen, which had continued to increase in size. Since middle of Dec. 1869 had suffered much from pain with numbness, shooting from right side of abdomen down along front and inside of thigh, and for one month this pain had been so severe as to keep him awake at night. For two years he had been steadily losing flesh, and had suffered at times from diarrhœa and hæmorrhoids. For six weeks before admission had kept his bed.

On admission, very emaciated; pains above referred to persistent. Pain down right thigh is increased when he lies on right side, and when he lies on left side there is an uncomfortable feeling of dragging about liver. Is most comfortable when he lies on back. Can retain beef-tea and bread in small quantity, but meat and most solid foods are rejected; vomiting comes on from one to several hours after meals. Tongue moist and white; no appetite; bowels have been costive for last six weeks, and have not been opened for four days. Hepatic dulness in r. m. l. 6½ in.; to right of middle line liver is smooth and not tender, but in left side of epigastrium is a hard, tender, nodulated mass, about size of large orange, apparently continuous with left lobe of liver, but slightly movable. Splenic dulness not increased. No jaundice, no ascites, and no swelling of legs. Pulse 84; thoracic organs healthy. Urine copious, clear, 1012, and contains neither bile-pigment nor albumen.

Under use of bismuth and hydrocyanic acid, and of creasote and subcutaneous injections of morphia and atropine, vomiting and pains were relieved; but there was no return of appetite, much flatulence and obstinate constipation; and patient continued to lose flesh and strength. On April 1 a nodule about size of pea could be felt beneath skin, an inch above umbilicus. On April 27 ascites was discovered; girth at umbilicus was 32 in., and on May 6 this had increased to 35 in. On May 2 feet were noticed to be swollen. On May 16 bile-pigment was found in urine, and stools were white; and on May 20 there was decided jaundice. On May 28 patient became much worse; he was so weak that he could scarcely turn in bed; tongue dry; paroxysms of dyspnœa on slight exertion. Towards evening he passed into a state of stupor, which continued until death early next morning.

Autopsy.—Eight pints of clear yellow serum in peritoneum. Liver of about normal size, but studded with masses of soft yellow cancer; its capsule thickened and adherent. The tumour felt during life was situated immediately below left lobe of liver, and sprang from duodenum and pyloric end of stomach. The first part of duodenum, to length of 5 in., had its coat greatly (⅔ in. at some places) thickened from cancerous deposit, at some places firm and translucent and at others softer, more opaque and yellow. This morbid deposit extended also four or five inches into stomach, but beyond pylorus it was not nearly so thick. Mucous membrane of first part of duodenum and of pyloric end of stomach extensively ulcerated, and channel through duodenum narrowed. On outer surface of duodenum was a large mass of cancerous excrescences. Glands in fissure of liver and mesenteric glands enlarged from cancerous deposit, and lumbar glands also enlarged so as to form a tumour pressing peritoneum forwards and eroding upper lumbar vertebræ; body of first lumbar vertebra almost eaten through. Spleen 5¼ oz. healthy. Kidneys, heart, and lungs healthy. Glands around root of left lung enlarged to size of hen's egg from cancerous deposit; ten ounces of fluid in left pleura.

CASE CXXIX.—*Cancer of Duodenum surrounding Hepatic Duct—Secondary Cancer of Liver—Jaundice and Ascites—Death from Lobular Pneumonia.*

Thomas M——, aged 36, carman, adm. into Middlesex Hosp. July 28, 1869. Father and mother alive and healthy; no history of malignant disease in family. Never had syphilis, and excepting contagious diseases of childhood and an attack of variola, had good health up to present illness. On Dec. 24, 1868, he got wet through and his clothes dried on him. Three days afterwards was seized with cough and a dull aching pain below right nipple. After three days pain shifted to epigastrium, where it remained nine days, but was never very severe, and during this time he vomited on two or three occasions. On pain ceasing, which it did rather suddenly, he noticed that skin and eyeballs were yellow. Since then he has been free from pain, nausea, or vomiting; but jaundice has persisted, there has been a constant feeling of general lassitude with moderate diarrhœa, and he has continued to lose flesh. Five weeks before admission his feet and legs began to swell, but he did not give up his work until five days before admission, when he was attacked with cough and expectoration.

On admission, patient was very emaciated and deeply jaundiced; considerable anasarca of both legs and decided pitting of trunk. Complained chiefly of great weakness and of diarrhœa; seven or eight motions in 24 hours; stools liquid, without any trace of bile-pigment, but depositing a granular slate-coloured sediment, containing apparently altered blood. Tongue clean, red and dry down centre; thirst; no vomiting. Liver enlarged; measures in r. m. l. 5 in., of which 3 in. below ribs; surface smooth, and not tender; lower margin firm and sharp. Slight ascites; no enlargement of abdominal veins or of spleen; no hæmorrhoids. Pulse 92, rather feeble; signs of heart normal. Frequent cough with expectoration of tenacious bronchial mucus; resp. 24; sonorous and sibilant râles with prolonged expiration over front of both lungs and a few moist sounds at back; no dulness. Temp. 101·4°. No itchiness of skin. Urine 1010; contains much bile-pigment, but no albumen.

Patient was ordered milk, eggs, 3 oz. of brandy, a mixture of bismuth, chloric ether, and a few drops of laudanum and sinapisms to chest. For first two or three days he felt much better, tongue became moist, and diarrhœa abated. The fever, however, persisted, pulse ranging from 88 to 120 and temperature from 101·4° to 104°. On night of August 1 he became much worse; respirations rose to 44; and on Aug. 2 he was very low and prostrate; scarcely conscious, dry tongue and involuntary evacuations. These symptoms continued until death in evening of Aug. 3.

Autopsy.—Much sero-sanguineous infiltration of both lungs; and in lower lobes of both organs (most in right) were many small scattered nodules of recent lobular pneumonia. Stomach healthy. Orifice of bile-duct surrounded by a mass of encephaloid cancer projecting into duodenum. A similar, but much smaller, growth projected from mucous surface a few lines further down. The larger growth, on dissection, was found to be about size of a walnut. Beyond it common and hepatic ducts were greatly dilated, former measuring 1¼ in. in circumference. Liver large, weighing 74½ oz., and containing several small, isolated, and at some parts confluent, nodules of morbid deposit, composed of a fibrous stroma enclosing in its meshes large ovate and caudate cells. Spleen 11 oz.; healthy. All other abdominal organs healthy.

The jaundice in the next case was also the result of cancer, the primary seat of which appeared to be in the areolar tissue and glands surrounding the head of the pancreas. But the immediate cause of the jaundice was an independent pendulous cancerous tumour growing in the interior of the bile-duct. Similar tumours were found in the portal vein and in the duodenum.

CASE CXXX.—*Cancerous Tumours of Duodenum, and in interior of Bile-ducts and Portal Vein—Jaundice—Ascites.*

Elizabeth M——, aged 50, adm. into St. Thomas's Hosp. Ap. 17, 1872. Her mother had a tumour in neck for 30 years, which nine months before death was said to 'turn into cancer,' and she died of the effects of it. Three years before admission patient noticed a lump about size of a hen's egg in right hypochondrium. Eight months ago this began to increase slowly, but it was never the seat of pain, except when pressed on; and her general health did not suffer until three months ago, when she began to lose flesh. Eight weeks ago she began to vomit food once or twice daily an hour or two after meals. Three weaks ago jaundice appeared; it was preceded for a week by diarrhœa, but by no pain nor increase of retching.

On admission patient was emaciated and deeply jaundiced, but countenance not expressive of pain. She complained of occasional attacks of severe pain in abdomen, lasting sometimes three hours and keeping her awake at night. A large prominence in centre of abdomen, mainly below umbilicus and due to a ventral hernia; but its upper part formed by a hard mass lying between umbilicus and right ribs, nodulated, sometimes tender, extending deeply backwards into abdomen, but at same time slightly movable and with a clear percussion space between it and liver. Hepatic dulness not increased. Tongue slightly coated; appetite fair, but uneasiness after food, which was often vomited about two hours after ingestion; bowels regular;

no bile in stools. No ascites. Urine deeply coloured with bile-pigment. Pulse 72. Heart and other organs healthy.

Patient was ordered a pill with creasote and morphia twice daily, and subsequently bismuth, and milk diet. At first there was a great improvement; but on May 13 appetite began to fail; and on May 16 pain was so severe that it was necessary to inject morphia subcutaneously, and there was some evidence of ascites, which rapidly increased. On May 19 slight diarrhœa set in, and motions were found to contain a large quantity of solidified fatty matter. From time that diarrhœa set in vomiting ceased for ten days. On June 4, abdomen was greatly distended with fluid, its walls being very tense and glistening, and patient complained much of tightness and dyspnœa. Legs also were becoming œdematous. Occasional vomiting of mucus streaked with blood. Sixteen pints of clear fluid were drawn off by paracentesis, after which patient was greatly relieved, but she continued to sink, and died on June 7.

Autopsy.—Nearly a gallon of deep yellow turbid serum in peritoneum. Behind duodenum was a large lobulated tumour composed mainly of enlarged lymphatic glands surrounding head of pancreas. Duodenum was found to contain a vascular pendulous mass of cancer, about 2 in. long and 1 in. broad, which broke off on opening bowel, but had been attached by a pedicle as large as a quill to interior of a sinus-like pouch 1 in. in length, close to, but distinct from bile-duct. Near to this were several other sinuses containing cylindrical masses of new growth. On cutting into tumour it was found to be traversed by sinuses containing similar masses of new growth. Similar new growths were found in interior of splenic and portal veins, as well as of branches of latter inside liver. Another was found inside hepatic duct at its junction with cystic. Beyond this, hepatic duct very greatly dilated, admitting two fingers; bile-ducts throughout liver also greatly dilated; gall-bladder greatly distended. Pancreatic duct could not be found. A small vascular growth was growing from peritoneal surface of fundus uteri. Spleen, kidneys, and other organs normal.

In Case CXXXI. the jaundice was caused by primary cancer of the glands in the portal fissure, which obliterated the hepatic duct but spared the common duct and the portal vein. Here also the patient at first improved and gained weight under treatment.

Case CXXXI.—*Scirrhus of Glands in Portal Fissure obliterating Hepatic Duct and causing Jaundice—Common Duct and Portal Vein free.*

William R——, aged 63, mattress-maker, adm. into St. Thomas's Hosp. April 7, 1875. An only child; father died young of scarlatina; mother died at 75. Twenty-four years ago, ill for six weeks with 'inflammation of lungs and liver,' and seven years ago again ill for six weeks with pain in back and diarrhœa. With these exceptions had enjoyed good health; never had gout or syphilis, and digestion had been good. Ten weeks ago began to lose appetite and to suffer from nausea, retching, and pains in back, shoulders, and stomach. After two weeks jaundice appeared, and at end of another week he was obliged to give up work. From first he had rapidly lost flesh and strength.

On admission, emaciation, deep jaundice; great itchiness, keeping him awake. Stools devoid of bile-pigment and urine loaded with it. Tongue white: appetite better than it had been; no vomiting for five days; bowels regular. No ascites; no enlargement of abdominal veins; and no induration around navel. Liver enlarged, measuring 6 in. in r. m. l.; surface smooth. No pain in liver; no œdema of legs; no albuminuria. Pulse 45 to 60; heart normal. Temp. 97°.

During first three weeks was treated with citrate of potash in effervescence, and improved considerably, gaining 2 lbs. in weight; but on two or three occasions he had an attack of rather severe pain across upper part of abdomen, lasting about an hour. On April 28 he had an unusually severe attack of pain with a return of vomiting; and after this appetite again failed; vomiting and pain recurred frequently; and there was a daily loss of flesh and strength. On June 11 he began to vomit black blood, and to pass a considerable quantity in clots per anum. After this he became rapidly weaker, and on June 20 he died.

Autopsy.—No fluid in peritoneum. Pancreas and duodenum adherent to under surface of liver; but both healthy. In portal fissure was a nodule of new growth, size of small orange, of firm consistence, adherent to surrounding organs by fibrous bands, and penetrating substance of liver for about an inch. This growth surrounded, infiltrated, and completely obstructed hepatic duct for about half an inch. Above this all branches of duct were greatly dilated, and filled with thin bile. Greater part of right lobe of liver was converted into a cavernous structure formed by the dilated ducts, with atrophy and induration of intervening tissues; some of these dilated ducts projected like cysts, size of peas, from surface. Gall-bladder contained about three drachms of bile; a probe passed readily from it through cystic and common bile-duct into duodenum, and these ducts were of normal size. Wall of portal vein opposite point of obstruction of hepatic duct was infiltrated to $\frac{1}{20}$ in. in thickness for about a third of an inch, but its lumen was not obstructed. On microscopic examination, new growth was found to have structure of scirrhus. A few other of the lymphatic glands in the portal fissure were enlarged and contained new growth; but there was no other deposit either in liver or in any other organ. Stomach, intestines, spleen, kidneys, and heart healthy. Œdema and hypostatic congestion of both lungs.

In Case CXXXII. jaundice resulted from the obstruction of the common bile-duct by a scirrhous mass originating in the post-peritoneal glands. The mode of commencement was unlike gall-stones or catarrh of the ducts; and primary cancer of the liver, which at first suggested itself, was negatived by the shrinking which followed the original enlargement of the liver. The most probable diagnosis seemed to be cancer of the head of the pancreas, but the post-mortem examination showed that the disease had originated in the post-peritoneal glands, and in connection with this it is interesting to observe that the prominent symptom throughout was pain in the back.

CASE CXXXII.—*Cancer of Post-peritoneal Glands obstructing Bile-duct and Portal Vein—Secondary Deposits in Liver and Lungs.*

Ellen F——, aged 60, adm. into St. Thomas's Hosp. May 18, 1875. Father died at 57 of typhus; and mother at 60; two brothers and one sister died young of variola. Married at 17; 12 children; habits temperate. Ten years ago had low fever; and two years ago some slight hepatic derangement; but with these exceptions health good till about six months ago, when she began to lose flesh and strength, and to suffer from loss of appetite, flatulence, and transient attacks of pain in back. After two months pains in back recurred more frequently; two months later nausea and itchiness of skin set in; one month before admission she began to be jaundiced. After jaundice appeared, pain in back became more constant and severe; ten days before admission she had for first time a sharp pain in liver lasting about a minute, and four days before admission she vomited once.

On admission, emaciated and very deeply jaundiced, with much itchiness of skin. Still much pain across back, but none in liver, which is much enlarged. Upper margin not too high, but lower margin fully 3½ in. below ribs in r. m. l., vertical dulness here measuring 6 in.; surface smooth, firm, not tender; no induration about umbilicus; no ascites; no œdema of legs nor enlargement of spleen. Tongue coated; breath offensive; appetite middling; no retching; much flatulence, but no severe pain after food; bowels regular; stools clay-coloured, and sometimes contain blood after straining. Urine loaded with bile-pigment; no albumen. Pulse 72; heart- and lung-signs normal.

Patient was treated with mineral acids, nux vomica, and ginger, with occasional aperients. She had fish or meat and a small quantity of wine. She improved at first, but then gradually got worse. Pain in back was very constant and was relieved by morphia; but no return of hepatic pain. On May 21, June 27, and July 22, she vomited food. At first bowels were costive, but on June 20 she had an attack of diarrhœa which lasted several days, five or six motions a day, and after this motions usually dark, as if from blood. Emaciation increased; jaundice persisted; liver gradually diminished in size; and on July 24 ascites was discovered, which rapidly increased. She was still able to eat and retain meat; but on Aug. 7 she became very prostrate and mind wandered, and on Aug. 9 she died.

Autopsy.—Peritoneum contained a large quantity of clear bile-stained fluid. Behind head of pancreas and projecting slightly above it was a dense nodulated mass, about size of small orange, firmly adherent to surrounding parts, and enclosing bile-duct and portal vein. On section this was found to have originated in glands behind pancreas, and to consist of dense cicatricial tissue. Superior mesenteric artery passed through it near its centre; it was compressed and narrowed, but its coats were free from infiltration. Common bile-duct passed into it at its upper part, about half an inch from junction of cystic and hepatic ducts, and became completely obliterated. Portal vein was also narrowed and its walls much thickened and infiltrated for about three quarters of an inch, and opposite point of entry into mass was an adherent discoloured thrombus, extending upwards for about an inch, but not completely obstructing vessel. Gall-bladder distended with a clear colourless viscid fluid and about sixty small calculi, size of peas; its fundus was about $\frac{3}{4}$ in. thick from presence of new growth, of which there were also several rounded nodules in adjacent substance of liver. Liver atrophied and dense; large patches of fibrous thickening of capsule; branches of hepatic duct greatly dilated and full of colourless fluid. Catarrhal inflammation and hæmorrhagic erosions of stomach. Spleen, kidneys, and heart healthy. Lower lobes of both lungs studded with numerous nodules of new growth similar to those in liver, from size of a mustard-seed to that of a large pea.

LECTURE XI.

JAUNDICE.

JAUNDICE INDEPENDENT OF OBSTRUCTION OF THE BILE-DUCT; DIAGNOSIS OF THE CAUSES OF JAUNDICE.

GENTLEMEN,—In my last lecture I explained to you that jaundice in those cases where it is independent of any mechanical impediment to the escape of bile from the liver might be referred to one of the following causes, viz.—

I. Poisons in the blood interfering with the normal metamorphosis of bile.

II. Impaired or deranged innervation interfering with the normal metamorphosis of bile, or increasing its secretion.

III. Deficient oxygenation of the blood interfering with the normal metamorphosis of bile.

IV. Excessive secretion of bile, so that more is absorbed than can undergo the normal metamorphosis.

V. Undue retention of bile in the biliary passages and bowels, from habitual or protracted constipation.

I purpose devoting the greater part of this lecture to the consideration of jaundice from these various causes.

I. JAUNDICE FROM POISONS IN THE BLOOD.

Cases are not uncommon in which jaundice results from a poisoned or morbid condition of the blood, such as that which exists in persons affected with the various specific fevers. It is very probable that when jaundice occurs in such cases, its mechanism is not always precisely the same. Sometimes, as for instance in many cases of ague and relapsing fever, it is associated with considerable enlargement and congestion of the liver, and this congestion is often the chief, if not the sole, cause of the jaundice; and at other times the duodenal orifice of the duct may be plugged by catarrhal inflammation. But in many, and these are much the more serious cases, the jaundice is independent of either congestion of the liver, or of obstruction of the bile-duct: during life there is no lack of bile in the evacuations, and after death the liver is often anæmic rather than congested. In these cases too, the cerebral and other phenomena of the typhoid state are usually prominently developed, and there is reason to believe that here, as in other diseases, this typhoid condition is due, not to the presence of bile in the blood (see p. 322), but to imperfect elaboration or elimination of the normal products of metamorphosis of the blood and tissues, of which the jaundice is only a visible sign. Although careful observations on this form of jaundice are still wanting, it has been repeately noticed that there is a diminution of urea in the urine, and that in some instances the urine has been found to contain leucin and tyrosin, which, as I have already told you, are indications of imperfect metamorphosis (see pp. 263, 324). The general condition of the patient, in fact, is very similar to what has been described to you in a previous lecture as occurring in acute or yellow atrophy of the liver (see p. 262), and it is very probable that the pathology of the two conditions is similar. In both there is a morbid condition of the blood, as the result of which the metamorphoses which usually go on in that fluid are impeded or arrested; and there is a deficient excretion of urea and a tendency to the development of leucin and tyrosin in the liver, spleen, kidneys, blood, and urine. In both, the jaundice is probably merely one of the results of this impaired metamorphosis, the bile-pigment absorbed into the blood not being transformed as in health. Occasionally the liver presents an appearance like that of an early stage of acute atrophy, and indeed I have already had occasion to tell you, that the poisons of typhus fever and of other allied diseases must be reckoned among the causes of yellow atrophy of the liver (p. 266).

We may now consider very briefly jaundice as it results from the several blood-poisons, some of which give rise to it much more readily than others.

1. *The Poisons of the various Specific Fevers.*

a. Yellow Fever.

The yellow fever of the tropics derives its name from the frequency with which it is complicated with jaundice. It has been demonstrated over and over again that the yellow suffusion of the skin and eyes in this disease is occasioned by the presence of bile, which is also found in the urine. Post-mortem examinations and the fact that in the earlier stages of the disease there is a full supply of bile in the alvine evacuations have satisfactorily proved that the jaundice is independent of any impediment to the escape of bile from the liver. On the other hand, as in acute atrophy of the liver, the jaundice is usually associated with hæmorrhages, 'black vomit,' delirium, and the other symptoms of the typhoid state. There are good reasons also for believing that this typhoid condition is due to the same cause as in acute atrophy, viz., impaired or deranged metamorphosis in the blood and tissues, and retention in the system of those products of metamorphosis which ought to be eliminated by the kidneys. The urine in most cases is albuminous and contains tube-casts, and is occasionally suppressed. Roche has found a deficiency of urea in the urine, but a large quantity of it in the blood;[1] Blair has detected a large amount of carbonate of ammonia in the blood, and also in the expired air;[2] while Lallemant describes the sweat as of a penetrating urinous odour.[3] The liver at first is enlarged from hyperæmia, but in the advanced stage of the disease it is pale and reduced in size, and the secreting cells are often loaded with oil.[4] The kidneys are also usually found to be large and congested in the early stage, but later in the disease the cortex is hypertrophied and the secreting tubes gorged with granular epithelium. From what has been stated it seems but fair to conclude, that the jaundice of yellow fever is only one of the results of that impairment or derangement of the metamorphoses taking place in the blood and tissues, of the existence of which there is such abundant proof.

b. Malarious Remittent and Intermittent Fevers.

The occurrence of jaundice in the malarious remittent and intermittent fevers of India, Algeria, and of other countries where true yellow fever is believed to be unknown, has been repeatedly noted. Twenty-four years ago I met with it in the malarious fevers of Burmah; [1] and Morehead, one of the latest and best writers on Indian diseases, observed jaundice in twenty-eight out of one hundred and fourteen cases of remittent fever.[2] In Algeria jaundice has been sometimes noted in as many as seven-tenths of the cases of intermittent fever.[3] The jaundice in these cases arises in different ways. Sometimes it is associated with congestive enlargement of the liver, or with gastro-duodenal catarrh, more or less obstructing the flow of bile and causing deficiency of bile in the motions; in both these cases the general symptoms are often mild. But in other cases which are usually fatal, jaundice is found associated with a dry, brown tongue, drowsiness, delirium, tremors, subsultus, and other symptoms of the typhoid state, with petechiæ and hæmorrhages from the stomach and bowels, and with albuminous and bloody urine, which is sometimes completely suppressed.[4] Careful analyses of the blood and urine in these cases have still to be made, but there can be little doubt that the general condition is similar to, if not identical with, that of the typhoid state in yellow fever, in British typhus, and, in fact, in acute diseases generally.[5] In these severe cases of remittent fever the bile-ducts have been found by Morehead and other observers perfectly patent and free from catarrhal inflammation, while the liver has been noted to be but slightly congested, and sometimes pale and in a state of fatty degeneration. The jaundice in these cases appears to result from a condition of the blood unfavourable to the metamorphosis of the absorbed bile.

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[1] Yellow Fever, Philadelphia, 1855.

[2] Report on Yellow Fever, by Daniel Blair, M.D., pp. 39, 40. Brit. and For. Med.-Chir. Rev., April 1856.

[3] Frerichs, op. cit. i. p. 183.

[4] Of thirteen fatal cases dissected by Louis at Gibraltar the consistence of the liver was diminished in seven. 'Its colour was altered in every case; sometimes it was of the colour of fresh butter, sometimes of a straw yellow, or of a clear coffee and milk colour, sometimes of a gum yellow, sometimes of an orange yellow.' Graves, Clin. Lect., 2nd ed. i. p. 283.

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[1] Notes on the Climate and Diseases of Burmah, Ed. Med. and Surg. Journ. April 1855, p. 229.

[2] Clinical Researches on Disease in India, 2nd ed. 1860, p. 73.

[3] Boudin, Traité des fièvres intermit. Paris, 1842.

[4] Frerichs, op. cit., vol. i. p. 180

[5] In 1853 I found no albumen in the urine of persons suffering from remittent fever in Burmah. My observations, which, for the most part, were made early in the disease before the supervention of typhoid symptoms, have been quoted as establishing a distinction between malarious remittents and true yellow fever. The comparative frequency, however, of albuminuria in yellow fever is probably due to the fact that the typhoid state is much more common in this disease than in malarious remittents. When the typhoid state is developed in remittent fever, it would indeed be extraordinary if it differed from the typhoid state of all other diseases in the absence of albuminuria. Moreover, in intermittents depending on the same malaria as remittent fevers, albumen and even blood are not uncommon in the urine.

c. *Relapsing Fever.*

Jaundice has been a frequent symptom in the relapsing fever of Great Britain and Ireland. Indeed, the frequency with which relapsing fever has been complicated with jaundice, and even with black vomit, has often caused it to be mistaken for true yellow fever In 1826 Drs. Graves and Stokes published an account of the 'yellow fever' of Dublin, and the twenty-first chapter of the first volume of Graves' unrivalled Clinical Lectures is entitled 'Yellow Fever of the British Islands.' It is now generally admitted, that the cases described by these writers were instances of relapsing or famine fever complicated with jaundice and cerebral symptoms, and their distinctness from true yellow fever was pointed out at the time by O'Brien.[1] The Scotch epidemic of 1843 was likewise regarded as closely allied to, if not identical with, yellow fever by many of its most distinguished observers, and it was even fancied that the disease had been imported into Glasgow by merchant-vessels from the West Indies, although in truth it had been prevailing in the east of Scotland for some time before it appeared in Glasgow.[2] There is, it is true, a strong resemblance between the more severe forms of relapsing fever complicated with jaundice and typhoid symptoms, and tropical yellow fever, but we have here another illustration of the mistakes which, I have told you, are apt to result from founding analogies or differences between acute specific diseases on symptoms alone, and of neglecting the circumstances under which they appear, or, in other words, their causes.

The frequency of jaundice in relapsing fever has been variously estimated, but on an average it may be said to have been noticed in one out of every five cases. The jaundice is independent of obstruction to the escape of bile from the liver. In many cases the associated symptoms are mild and the patients recover, and then the jaundice is probably the result of the congested condition of the liver which is so common in relapsing fever. Yet most observers of relapsing fever have agreed in making jaundice a formidable symptom, and it has certainly been often accompanied by hæmorrhages, including black vomit, a dry brown tongue, delirium, coma, subsultus, convulsions, and other cerebral symptoms; and at the same time it has not necessarily been associated with hepatic congestion, but often with a soft, pale, and yellow condition of the liver and with the presence of leucin and tyrosin.[1] As in true yellow fever, however, these serious symptoms are not due to the presence of bile in the blood; but the jaundice is only an outward and visible sign of important changes in the blood interfering with the natural metamorphoses. The urine has been ascertained to be suppressed or diminished in quantity and very deficient in urea, which has been found in abundance in the blood and in the cerebral fluid.[2]

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[1] Trans. Queen's Coll. of Phys. of Dublin, 1828, p. 532.

[2] Murchison on the Continued Fevers of Great Britain, 2nd ed. 1873, pp. 47, 395.

d. Typhus Fever.

Very opposite statements have been made as to the occurrence of jaundice in true typhus fever. Sir W. Jenner states that he has never met with it, whereas, according to Frerichs, several epidemics of petechial typhus have been characterised by the frequency of jaundice. It is probable that Frerichs has been misled by the frequency with which epidemics of typhus and relapsing fevers have prevailed together, and by the fact that in most instances the latter disease has been regarded as a mere variety of the former. At all events, in this country and in Ireland jaundice is a very rare complication of true typhus. In 1843 Dr. Henderson referred to the occurrence of jaundice in typhus fever;[3] two cases are recorded by Frerichs;[4] fifteen observed by myself are referred to in my work on the Continued Fevers of Great Britain; and Dr. Hudson of Dublin also speaks of jaundice as 'a very rare complication in typhus.'[5] The rarity of jaundice in typhus, as well as the severity of the cases in which it occurs, may be judged of from what has been observed at the London Fever Hospital. Out of 7,604 cases of true typhus admitted into the hospital during the years 1862, 3,

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[1] In two fatal cases of relapsing fever with jaundice, hæmorrhage, and typhoid symptoms, recorded by Sir J. Rose Cormack, the liver was found in one 'of the natural colour and consistence,' and in the other it was 'softer than natural,' and 'the section exhibited a dingy lightish colour.' Nat. Hist., Path., and Treatment of the Epidemic Fever at present prevailing in Edinburgh &c., 1843, Cases VII. and VIII. In the epidemic of relapsing fever which occurred in St. Petersburg in 1864, the liver was repeatedly found in a state of acute atrophy, and in two cases of this sort Zuelzer detected in the organ crystals of leucin and tyrosin.

[2] See the evidence on this matter collected in my work on Fevers, 2nd ed. p. 367.

[3] Edin. Med. and Surg. Journ. 1844, vol. lxi. p. 220.

[4] Op. cit. i. pp. 168, 170.

[5] Lectures on the Study of Fever, 1867, p. 88.

4, and 5, jaundice was noted in only 16, or once in every 475 cases. Of the 16 cases, 12 were fatal, and deducting 2 cases where the jaundice did not occur until convalescence and was evidently catarrhal, of the remaining 14 patients in whom jaundice coexisted with the typhus rash 12 died. As in the specific diseases already mentioned, the jaundice is not due to any obstruction of the bile-duct; it is likewise independent of hepatic congestion. The hepatic tissue in the cases which I have had an opportunity of dissecting has been preternaturally pale and soft, all trace of division into lobules has in some instances disappeared, and the secreting cells have contained a large quantity of oil and have appeared to be undergoing disintegration, while both Frerichs and myself have found leucin and tyrosin in both the hepatic and renal tissue, and also in the urine.[1] In one of my cases (Case CXXXIII.) also it was ascertained that, as in yellow atrophy (see p. 263), urea had almost disappeared from the urine. Excepting the presence of jaundice, there is nothing very remarkable in these cases of typhus. Typhoid symptoms are always present in a prominent degree, and, as I have elsewhere endeavoured to show, these symptoms are probably due to an imperfect elaboration and retention in the system of those products of blood- and tissue-metamorphosis which ought to be eliminated by the kidneys. Convulsions, which may be regarded as the acme of the typhoid state, are now acknowledged to have a uræmic origin in typhus as well as in scarlatina, and I have placed on record cases of typhus,[2] without as well as with convulsions, in which urea has been found in the serum of the blood. When jaundice then occurs in typhus, it does not account for the other serious symptoms with which it is usually associated, nor does it, in itself, probably contribute in any way to the fatal event; it is merely one indication of an unusual impurity and derangement of the normal metamorphosis of the blood, as the result of which the absorbed bile is not transformed as in health, or even as in ordinary cases of typhus.

Occasionally jaundice in typhus admits of another explanation than that now offered. Its appearance in Case CXXXV. was probably determined by the double pneumonia, and in Case CXXXVI., where it occurred during convalescence, it seemed due to a condition approaching that of pyæmia, rather than to be a direct result of typhus.

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[1] Murchison, Continued Fevers of Great Britain, 2nd ed. 1873, p. 210.

[2] See my work on Fevers, pp. 161, 174.

e. Enteric or Pythogenic Fever.

I have met with jaundice in only four cases of enteric fever, of which three were fatal. In one (Case CXXXVII.) which recovered, it occurred during a relapse of the fever, and was probably due to catarrh of the bile-duct; in a second (Case CXXXIX.), it appeared on the fourteenth day and was associated with albuminuria, and during convalescence with thrombus of the femoral veins; the albuminuria persisted, and the patient died within six months. In the remaining two cases the jaundice occurred during the primary fever; both were fatal; and in both the liver after death was found to be small, and its secreting cells loaded with oil. In a fifth case communicated to me (Case CXXXIX.), the jaundice came on towards the termination of a severe attack and persisted through convalescence. A case is recorded by Andral where the jaundice was noted at the commencement of the third day, and where the patient died on the ninth day of pneumonia of the left lung.[1] Louis has recorded two fatal cases, one of which was associated with parotid bubo and secondary purulent deposits in the liver, and the other with erysipelas of the leg.[2] Sir W. Jenner never met with jaundice in enteric fever, but refers to a preparation from a fatal case which occurred on the west coast of Africa. Two fatal cases are recorded by Frerichs.[3] In one the jaundice did not appear until the thirty-seventh day, when the patient appeared to be convalescent, and on the forty-first day the patient died with symptoms of pulmonary œdema; the urine was scanty, and after death the kidneys were found to be congested, while leucin and tyrosin were discovered in the hepatic tissue. In the other the jaundice appeared as early as the fifth day, and was accompanied by profuse epistaxis and violent delirium; death occurred on the eighth day before the commencement of ulceration in the ileum, and the liver was found to be in a state of acute yellow atrophy. Jaundice appears to be on the whole a rarer symptom in enteric fever than in typhus, and of the few cases where it occurs in some the jaundice is probably due to catarrh of the bile-duct. In others, however, its pathology appears to be the same as that of jaundice in the specific diseases already considered.

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[1] Clinique Médicale. 3me éd. 1834, tom. i. p. 10.

[2] Recherches sur la Fièvre typhoïde, 2me éd. Paris, 1841, Obs. 17 and 26.

[3] Op. cit. vol. i. pp. 172, 215

f. Scarlatina.

You will find in Dr. Graves's Clinical Lectures (vol. i. p. 453) two cases of scarlatina referred to which were complicated with jaundice and enlargement of the liver and ascribed to hepatitis, and that a chronic form of hepatitis is spoken of as a common sequel of scarlet fever. Dr. G. Harley has also related a case of scarlet fever complicated with jaundice from what was believed to be congestion of the liver.[1]

From my own experience I am led to the conclusion that jaundice in scarlet fever is extremely rare. Out of about 2,000 cases that came under my care prior to 1868, it occurred in only five. Three of the five cases were fatal. In two of the fatal cases an autopsy was performed; in one the liver was not congested in the slightest degree, but was pale and fatty (Case CXLI.); in the other (Case CXL.) it presented a nutmeg appearance, the margins of the lobules being pale and their centres full of blood; in both cases the bile-ducts were perfectly patent; the urine in both cases contained albumen, but it is to be regretted that no examination was made for leucin or tyrosin. It is very probable that when jaundice appears in scarlet fever it may be sometimes due to hepatic congestion or to catarrh of the bile-ducts; but in other cases, and these are the more fatal, it is evidently independent of congestion or of obstruction of the ducts, and is probably the result of serious derangements in the metamorphoses of the blood.

g. 'Epidemic Jaundice.'

Most writers on jaundice refer to its occasional occurrence in the epidemic form. You will find an account of several epidemics of jaundice in Frerichs's work on the Liver.[2] These epidemics have varied greatly in their fatality, and probably also in their nature. In some, not a single patient has died. This was the case in an epidemic at Chasselay, referred to by Frerichs, where the jaundice commenced with gastric catarrh and the stools were always pale. A similar observation was made at Pavia in 1859. Of 1,022 French troops stationed there, 71 were attacked with jaundice, but all recovered; the cases were characterised by pain in the epigastrium and hypochondria and by enlargement of the liver and spleen; and the epidemic was attributed to marsh miasmata, conjoined with unusual heat, fatigue, and intemperance.[1] On the other hand, in an epidemic which prevailed at Essen in 1772, which attacked chiefly children, assumed an intermittent type, and was characterised by delirium and other nervous symptoms, a large proportion of the patients perished. In another epidemic which occurred in the island of Martinique in 1858 the disease was extremely fatal among pregnant females; of 30 women attacked at St. Pierre during pregnancy 20 aborted and died, death being preceded by delirium, coma, and other symptoms closely resembling those of acute atrophy of the liver.[2] In 1862 a remarkable epidemic of jaundice occurred at Rotherham, where the condition of the drainage was notoriously bad. In the autumn of that year Rotherham was visited by a very fatal outbreak of enteric fever. This was followed by an epidemic of jaundice early in the following year, and in the month of February it was stated that not fewer than 150 persons were suffering from it, but that none who had passed through the fever in the previous autumn had been attacked.[3] According to Sir Thomas Watson, jaundice was epidemic in London in 1846, just after the prevalence of extremely hot weather,"[4]—a season which was also remarkable for an unusual prevalence of enteric fever.

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[1] Pathology and Treatment of Jaundice, p. 93.

[2] Op. cit. i. p. 188.

Most of these epidemics seem to have been due to some malarious poison (see *antea*, p. 154); some perhaps may have resulted from a chill or other atmospheric influence. The mechanism of the jaundice has probably varied with the severity of the epidemic. Sometimes it appears to have been occasioned by congestion of the liver or catarrh of the bile-ducts, but in others where it was accompanied by delirium and typhoid symptoms and was extremely fatal, and where the whole phenomena bore a close resemblance to those of acute or yellow atrophy of the liver, it has more probably been due to morbid conditions of the blood interfering with the normal metamorphoses. I have already told you that jaundice which in the first instance appears purely catarrhal may terminate fatally with symptoms of acute atrophy (p. 267).

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[1] Medical Times and Gazette, June 8, 1861, p. 607.

[2] Brit. Med. Journ. Feb. 7, 1863.

[3] Lancet, 1863, vol. i. pp. 222, 374.

[4] Op. cit. 5th ed. ii. 683.

2. *Animal Poisons.*

a. Pyæmia.

In a large proportion of cases of pyæmia, whether from external wounds or injuries, from parturition, or from internal causes (see p. 166), there is jaundice of the skin, conjunctivæ, and urine. Many cases of this sort came under my notice in the London Fever Hospital. The jaundice usually commences early in the disease and continues to increase till death; but it is rarely intense, and sometimes it is so slight that it is apt to be overlooked. The bowels are usually relaxed and the evacuations contain plenty of bile. Occasionally, as I have explained to you in a former lecture (see p. 164), the liver is found to contain purulent deposits, but in most cases nothing can be detected in it to account for the jaundice. The organ is pale and anæmic, and the bile-ducts are patent and free from inflammation (see Case CXLIV.).[1] The urine, in addition to bile-pigment, often contains albumen or blood, indicating a condition of the kidneys unfitting them for eliminating the large quantity of urea which is manufactured in pyæmia in common with other pyrexial diseases. In most cases of pyæmia the tongue after a time becomes dry and brown, and there are more or less stupor and delirium, and in fact all the phenomena of the typhoid state met with in typhus and in other diseases. The abnormal condition of the metamorphic processes going on in the blood and the accumulation in the blood of the products of metamorphosis which ought to be eliminated by the kidneys, to which this typhoid state is due, lead also to an impaired consumption of the bile which has been absorbed into the blood and account for the jaundice.

b. Snake-bites.

Since the days of Galen it has been known that the bites of snakes and vipers occasionally cause jaundice. The jaundice may be intense, and what is very remarkable is the rapidity with which it is sometimes developed. Speaking of cases of this sort, Dr. Mead long ago observed, 'intra non integram horam fit flavus, quasi ejus qui ictero laborat.'[1]

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[1] Virchow has maintained that the jaundice in pyæmia (as well as in typhus) is catarrhal, and due to a plug of viscid mucus in the duodenal orifice of the duct (Virchow's Archiv, 1865, xxxii. Hft i.). According to Frerichs, however, 'the bile-ducts are open and usually pour out a little thin secretion,' and this coincides with my own experience. Moreover, the fact that the stools always contain bile and that the jaundice is in most cases slight are opposed to this being the result of mechanical obstruction of the duct.

Careful records of post-mortem examinations in cases where death has been due to snake-bites are still wanting, but it is clear that the jaundice is independent of any obstruction of the gall-duct, as the vomited matters and stools always contain bile. The very rapidity also with which the jaundice is developed is opposed to its immediate cause being congestion of the liver, and suggests the idea that it is the result of disordered innervation, whereby there is induced an abnormal condition of the metamorphic processes going on in the blood. The general symptoms resulting from snake-bites,—viz. a quick small, irregular pulse, tendency to fainting, bilious vomiting, difficult breathing, cold perspiration, dulness of vision, derangement of the mental faculties and sometimes convulsions,[2] all point to serious derangement of the nervous system.

3. *Mineral Poisons.*

a. Phosphorus.

During the last few years numerous cases have been recorded both in this country and on the Continent of acute poisoning by phosphorus,[3] which are remarkable in this respect, that in almost every instance jaundice has been one of the symptoms noticed. I do not refer here to those cases of chronic poisoning by phosphorus where there is necrosis of the jaw, so common in persons engaged in the manufacture of lucifer-matches, but to cases where acute symptoms have followed one large dose of the poison. There has been much discussion as to what is the pathology of the jaundice in these cases. Virchow and other observers maintain that it is due to obstruction of the duodenal end of the bile-duct by thickening of the mucous membrane and a plug of mucus, and that although the stomach and duodenum have often been found to present no redness nor obvious sign of inflammation, there is nevertheless a 'cloudy swelling' of the gastric glands and thickening of the whole membrane.[1] Dr. O. Wyss, however, has shown that when dogs with a biliary fistula were poisoned with phosphorus jaundice was produced, which could not therefore be due to obstruction of the intestinal portion of the common bile-duct. After the appearance of jaundice much less bile escaped by the fistula, and that little was mixed with colourless mucus; sometimes mucus alone escaped.[2] This observation is interesting in connection with the fact, that almost all the descriptions of the post-mortem appearances agree in stating that the liver is in an extreme state of fatty degeneration, and that, as in acute atrophy, the secreting functions of the organ have been in a great measure abrogated. The appearance of the liver, in fact, has in many instances resembled very closely that of yellow atrophy. The symptoms moreover of acute phosphorus-poisoning—drowsiness followed by violent delirium, convulsions and coma, vomiting, albuminous or bloody urine, in which C. Schultzen[3] has discovered a substance closely allied to tyrosin, and a fluid condition of the blood with petechiæ and hæmorrhages—are utterly unlike those of catarrhal jaundice, and so closely resemble those of acute atrophy of the liver, that it has even been suggested that many of the recorded instances of acute atrophy have been really cases of phosphorus-poisoning.[4] It seems probable therefore that the jaundice of phosphorus-poisoning has a blood origin, and, like that of yellow fever and typhus, must be ascribed to an abnormal condition of the metamorphoses in the blood.

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[1] Tentamen de Vipera, p. 36.

[2] Christison on Poisons, 1829, p. 470.

[3] An account of many of these cases will be found in the Year Books of the Sydenham Society, 1859, p. 445; 1860, p. 440; 1861, p. 409; 1862, p. 428; 1863, p. 404; 1864, p. 423; and Biennial Retrospects, 1865–6, p. 434; 1867–8, p. 448; 1869-70, p. 453. Two cases are also recorded in the Fiftieth volume of the Medico-Chirurgical Transactions by Drs. Habershon and Hillier.

b. Mercury. c. Copper. d. Antimony.

The preparations of mercury, copper, antimony, and other irritant poisons have been known to cause jaundice, but only in exceptional cases. The mode of production of the jaundice has not been well ascertained, but the most probable explanation is that it is caused by the inflamed and swollen state of the mucous membrane blocking up the duodenal orifice of the bile-duct.

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[1] Archiv f. path. Anat. u. Phys. xxxi. p. 399.

[2] Archiv der Heilkunde, 1867, p. 419.

[3] Ueber acute Phosphorvergiftung und acute Leberatrophie, Berlin, 1869, pp. 29, 32, 36.

[4] See for instance references in Syd. Soc. Year Book for 1862, pp. 428, 430, and for 1863, p. 404. The points of differential diagnosis which H. Köhler (Schmidt's Jahrb. No. 147, p. 148) has drawn between acute atrophy of the liver and phosphorus-poisoning will, I am satisfied, not always hold good at the bedside.

4. *Chloroform and Ether.*

Chloroform and ether, according to Frerichs,[1] occasionally cause jaundice, while several observers have found that under their influence sugar passes off in the urine. The concomitant symptoms of jaundice from these substances are little known, and the cases are extremely rare, for after considerable search I have been unable to find the records of any. Most probably the jaundice has a blood origin, but its precise mode of production has still to be determined.

5. *Acute Atrophy of the Liver.*

In a former lecture (see pp. 260, 270) I have shown you that the jaundice in that remarkable disease, acute or yellow atrophy of the liver, is independent of obstruction of the bile-ducts, and that it is probably the result of some abnormal condition of the blood. The motions during life usually contain bile, and after death the gall-ducts are found to be perfectly patent, while on the other hand all the phenomena of the disease approximate it to those maladies which are known to result from some poison, such as typhus, enteric fever, pyæmia, and phosphorus-poisoning. I need only recall to your recollection the fact, already adverted to in this lecture, of leucin and tyrosin being found in the jaundice of typhus in common with that of acute atrophy, and the circumstance of the liver being found in a state of acute atrophy in a case of enteric fever complicated with jaundice. It becomes a question, indeed, whether the condition of the liver in acute atrophy be the cause of all the formidable symptoms with which it is associated, or whether it be not rather merely one of the consequences of some general disorder of the system, like that which is produced by many poisons. On a former occasion I told you that it had been repeatedly observed, that several of the residents in the same house had been attacked with acute atrophy almost simultaneously (p. 266). There is good reason also for believing that some of the instances of 'epidemic jaundice' have been examples of acute atrophy. In the epidemic, for instance, which prevailed in the island of

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[1] Op. cit. vol. i. p. 160.

Martinique in 1858, the jaundice was accompanied by delirium, coma, and other symptoms of acute atrophy, and, as in acute atrophy, the disease was especially frequent and fatal in pregnant females, who aborted before death. It is a matter for investigation whether the anatomical changes which are so notably present in the liver in cases of acute atrophy are really limited to that organ. Wagner, who is of opinion that many of the recorded instances of acute atrophy were probably cases of acute poisoning by phosphorus, on the ground of their complete clinical and pathological analogy with cases known to be of this nature, has drawn attention to the almost universal infiltration of every tissue of the body with oil in cases where death has been due to phosphorus, whereas this change had been previously recognised only in the liver. He found minute fat-granules in the epithelium of the kidneys, in the parenchyma of the lungs, and in the muscular fibres of the voluntary muscles and of the heart. These observations have been confirmed by other investigators, and while Bucquoy,[1] Buhl,[2] and Steiner[3] have discovered in the brain a fatty destruction similar to that which is found in the liver, kidneys, and heart, one cannot fail to be struck with the analogy which, in this respect, cases of phosphorus-poisoning bear to typhus, in which, as we have found, jaundice with leucin and tyrosin is apt to be developed. It is now well known that a granular degeneration of the voluntary muscles of the heart and of the renal epithelium is among the most common post-mortem appearances in typhus, and probably in most diseases where death has been preceded for some time by the typhoid state. Speaking of the kidneys in acute atrophy of the liver, Frerichs observes:[4] 'I have found the glandular epithelium infiltrated with granules, and in most cases in a state of fatty degeneration, and the tissue itself flabby and shrivelled.' Frerichs also speaks of 'a flabby shrivelled character of the muscular tissue of the heart,' and states that, 'in some cases the cerebral substance has appeared softened,' although he expresses doubts whether this condition was the result of commencing putrefaction, or a product of disease. There seems reason then for believing that the condition of the liver in acute atrophy is only one of many similar changes taking place throughout the body, as the result of some blood-poison. Trousseau indeed maintains that the symptoms of acute atrophy (malignant jaundice) can exist without any lesion of the liver, which cannot therefore be the cause of the change in the blood.[1]

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[1] Union Médicale, 1863, No. 81.

[2] Zeitschrift für rat. Med., 1852.

[3] Compend. der Kinderkrankheiten, 1873, 304.

[4] Op. cit. i. 227.

Dr. Grainger Stewart has advanced our knowledge a step further by recording cases showing that acute atrophy of the kidneys may not only coexist with acute atrophy of the liver, but that the morbid process in the kidneys may precede that in the liver.[2]

6. *Cirrhosis and Chronic Atrophy of the Liver.*

In a former lecture (p. 281) I have pointed out to you that in the advanced stages of cirrhosis of the liver, jaundice associated with cerebral symptoms and hæmorrhages is not uncommon. The motions in these cases are usually well-coloured with bile, and the pathology of the jaundice is probably the same as that of acute atrophy. The jaundice is usually slight, gradual in its onset, and attended, rarely by pain, but often by ascites.

II. IMPAIRED OR DERANGED INNERVATION INTERFERING WITH THE NORMAL METAMORPHOSIS OF BILE OR INCREASING THE SECRETION.

That jaundice may have a nervous origin has long been known. There are numerous instances on record of its being produced by severe mental emotions, such as fits of anger, fear, shame, or great bodily suffering. Concussion of the brain has been known to have a like effect. Villeneuve relates the case of a young soldier, who, being insulted in public, drew his sword and would have rushed upon the aggressor, but was restrained by the bystanders; in his vain efforts to wreak his vengeance, he became suddenly jaundiced; soon afterwards delirium set in and he died in convulsions. He also quotes the case of a young abbé, who, owing to a sudden fright from a mad dog breaking its chain and rushing at him, uttered a loud cry, fell down unconscious, and was taken up as yellow as saffron.[3] Mr. North witnessed a case in which an unmarried female, on its being accidentally disclosed that she had borne children, became in a very short time yellow; and a young medical friend of Sir Thomas Watson had an attack of intense jaundice, which could be traced to nothing else than his great and needless anxiety about an approaching examination before the Censors' Board of the College of Physicians.[1] There are two circumstances worthy of note in these cases:—1. The rapidity with which the jaundice is developed, the skin and conjunctivæ become yellow almost in a moment, and even before the appearance of any bile-pigment in the urine; and, 2. That cerebral symptoms, such as delirium, coma, and convulsions, often supervene upon the jaundice, and that the cases are then often fatal. These characters seem incompatible with the supposition that the jaundice can result from any mechanical obstruction of the bile-duct, or even from congestion of the liver, and make it more probable that it is caused by some derangement, through nervous influence, of the natural metamorphoses in the blood. In a former lecture I have told you that the 'pathemata mentis' constitute one of the causes of the general morbid state of which acute atrophy of the liver is one of the local manifestations.

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[1] Clin. Lect., Syd. Soc. Ed. iv. 312.

[2] Bright's Diseases of the Kidneys, 1868, p. 159.

[3] Dict. des Sciences Méd., 1818, Art. Ictère, p. 420.

It is very probable, however, as Dr. Bence Jones has pointed out,[2] that jaundice has occasionally a nervous origin of another sort. The circulation and secretion of all glands are controlled by the nerves which supply them. Claude Bernard has shown that if the sympathetic filaments of the sublingual gland be tetanised, the blood in the gland becomes very dark, and the saliva scanty and concentrated; but that if, on the contrary, the chorda tympani alone be tetanised, the blood in the gland presents an arterial hue, and the saliva is increased, though it contains a small proportion of solid matter. Similar results would no doubt be produced in the liver. Irritation of the sympathetic or paralysis of the branches of the pneumogastric nerve would probably contract the small blood-vessels and diminish the secretion of bile, while paralysis of the sympathetic, or irritation of the pneumogastric, would relax the capillaries and increase the rapidity of the circulation through the liver and the secretion of bile. Under these circumstances jaundice would be produced in the way which I shall explain to you presently when speaking of jaundice from congestion.

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[1] Lect. on Pract. of Physic, 5th ed. vol. ii. p. 682.

[2] St. George's Hospital Reports, 1866, vol. i. p. 193.

III. DEFICIENT OXYGENATION OF THE BLOOD INTERFERING WITH THE NORMAL METAMORPHOSIS OF BILE.

Whatever interferes with a due supply of oxygen to the blood interferes with those metamorphoses which in a healthy condition of the body are constantly taking place in it, and may thus impede or arrest the normal transformation of the absorbed bile and cause jaundice. It is deficient oxygenation which probably in a great measure accounts for many of the cases of true jaundice with bile in the stools (see *antea*, pp. 312, 347) met with in new-born infants. According to Dr. West, 'in the Dublin Lying-in Hospital, where the children are defended by the most watchful care from the evils either of cold or of a vitiated atmosphere, the occurrence of infantile jaundice is rare: while in the Foundling Hospital in Paris jaundice is so common that comparatively few infants escape it. Almost all the children at the Foundling Hospital have been exposed to the action of cold while being brought to the institution, and suffer from the combined influence of cold and bad air while inmates of it—causes which interfere very seriously with the due performance of the functions of the skin and of the respiratory organs.'[1]

The jaundice which occasionally accompanies acute pneumonia in the adult may possibly have a similar origin. In the course of acute pneumonia the skin and conjunctivæ occasionally become jaundiced, and bile-pigment appears in the urine without any disappearance of bile from the motions. The pneumonia in these cases is far from being always in the lower lobe of the right lung, as some writers have stated. Of 19 cases observed by Drasche, the inflammation was in the right lung in 7—in 5 at the base, in 1 at the apex, and in 1 in the entire lung; in 8 the left lung alone was affected; and in 4 the pneumonia was double.[2] The jaundice in these cases is independent of any obstruction of the bile-duct, for, as a rule, there is no lack of bile in the evacuations from the bowels. Various explanations have been offered as to its mode of production. It has been attributed to congestion of the liver from impeded circulation through the lungs, and possibly hepatic congestion may also be induced by nervous irritation reflected through the pneumogastric nerve from the lungs to the liver. Dr. Bence Jones has recently suggested that the jaundice in these cases is the result of an arrest of oxydation in the blood. These explanations, however, are not applicable in all cases. In some of the worst cases, the proportion of the lungs implicated in the inflammation is comparatively slight, and the jaundice is associated with typhoid symptoms and albuminuria. The urine in these cases is often of a bright red colour, but, what is remarkable, it is said not always to present the ordinary reaction of bile-pigment with nitric acid. The absence of bile-pigment from the urine has accordingly been regarded as an unfavourable symptom in icteric pneumonia. Of 14 cases in which bile-pigment was found in the urine by Drasche, only 3 died; but of 5 where the urine contained no bile-pigment 2 died.[1] It is probable that in these cases the jaundice has a similar blood-origin to that of typhus, pyæmia, &c., which we have already considered.

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[1] Lect. on Dis. of Infancy and Childhood, 5th ed. 1865, p. 602.

[2] Œsterrh. Zeitsch. f. prakt. Heilk. 1860, No. 23.

Deficient oxygenation of the blood from inhaling a vitiated atmosphere in badly-ventilated or overcrowded apartments in many instances no doubt induces 'bilious headaches' and functional derangement of the liver, and may even conduce to the development of jaundice.

IV. EXCESSIVE SECRETION OF BILE, MORE OF WHICH IS ABSORBED THAN CAN UNDERGO THE NORMAL METAMORPHOSIS.

If we can suppose that in a particular individual the oxygenation and other processes of metamorphosis going on in the blood are just sufficient to transform the whole of the absorbed bile, it is not difficult to understand that in the event of the quantity of bile being increased, part of it might not be transformed and jaundice would be the result. Now this is probably what actually takes place in cases of congestion of the liver. The vessels of the liver are distended, and the diffusing surface of the walls is consequently increased, and more than the normal quantity of bile is taken up into the blood. In many cases of congestion of the liver the quantity of bile secreted is also increased. This then appears to be the pathology of the jaundice from congestion of the liver. There is no obstruction of the bile-ducts, unless there be concurrent inflammation of the duodenum or ducts (see p. 132), and sometimes, indeed, there is bilious diarrhœa. If the bowels be constipated, the jaundice from congestion of the liver will probably be increased, as the bile instead of being cleared away will accumulate in the biliary passage, and will be absorbed in all the larger quantity by the distended vessels. Mercury, podophyllin, and other purgatives do good in these cases by sweeping away the bile as fast as it flows into the duodenum, and perhaps also by stimulating the gall-bladder and bile-ducts to contract through reflex action. As I have formerly told you (pp. 135, 330), there is no evidence that they stimulate the liver to increased secretion. If they did they would be injurious rather than otherwise in cases of jaundice from hepatic congestion.

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[1] Œsterrh. Zeitsch. f. prakt. Heilk. 1860, No. 23.

The symptoms, varieties, and causes of hepatic congestion have been already considered in a former lecture (see p. 131).

V. UNDUE ABSORPTION OF BILE INTO THE BLOOD FROM HABITUAL OR PROTRACTED CONSTIPATION.

I have already explained to you one way in which jaundice may result from constipation, viz., from the pressure of fæces accumulated in the colon upon the bile-duct. Independently of causing pressure, however, it is very probable that a sluggish state of the bowels often contributes to the development of jaundice, partly by impeding the portal circulation and inducing congestion of the liver, and partly by causing an accumulation of bile in the biliary passages and duodenum, and thus favouring its absorption into the blood. It is under these circumstances that there is often developed the condition known as 'biliousness from a torpid liver,' where the patient suffers from languor, headache, furred tongue, flatulence and constipation, a feeling of weight and oppression after meals, and not uncommonly hypochondriasis; and, although these symptoms may last a long time without actual jaundice, this is liable to supervene at any time from irritating ingesta, or from other causes, which increase the congestion of the liver. The liver in these cases, so far from being 'torpid,' is perhaps secreting too much bile, while mercury and other purgatives do good, not as is generally supposed, by stimulating the liver to increased secretion, but by getting rid of a great portion of the bile as fast as it is thrown out, and thus preventing its absorption.

Treatment of Jaundice independent of Obstruction of the Bile-duct.

The treatment of jaundice independent of obstruction of the bile-duct must be regulated according to its cause.

1. In the jaundice from constipation, or in the state of 'biliousness' already adverted to which is short of actual jaundice, purgatives must be given in the first place, and of these the best are occasional doses of calomel, blue pill, or podophyllin, with salines, such as the sulphates of soda, potash and magnesia, the citrate of magnesia, seidlitz powder and the bitartrate of potash, or the mineral waters of Friedrichshall, Püllna, or Carlsbad. Alkalies and their salts with the vegetable acids are also useful, partly in correcting acidity of the stomach, but mainly by carrying off by the kidneys the products of blood- and tissue-metamorphosis, the presence of which in the blood is the probable cause of the languor and other symptoms from which the patient suffers. At the same time fermented liquors, wines, spices, fat, and all rich or indigestible articles of diet, which are calculated to irritate or congest the liver, must be forbidden. These are the measures most likely to relieve the jaundice or 'biliousness' resulting from constipation. But the great object of the practitioner in all such cases ought to be so to modify the patient's habits and diet as to secure if possible a regular action of the bowels without the necessity of constantly having recourse to medicine. It is always well to enjoin regular exercise in the open air and the use of brown bread, or of such articles of diet as the individual knows from experience to have an aperient effect upon the bowels; and in cases where purgatives give only temporary relief, more permanent benefit may be expected from purgative mineral waters, such as those of Carlsbad, Friedrichshall, Harrogate, Cheltenham, or Leamington. When the constipation and its immediate effects have been relieved, but where the patient still suffers from weakness and symptoms of atonic dyspepsia, the mineral acids in conjunction with the vegetable bitters, such as nux vomica, quinine, gentian, or cascarilla, or with pepsin, may be expected to do good; while the bowels are kept open by a daily pill of aloes, nux vomica, and soap.

2. The treatment which is appropriate in cases of jaundice dependent upon congestion of the liver we have already considered, when treating of congestion as one of the causes of

enlargement of the organ (see p. 134). It is necessary to remember that some of the cases of jaundice occurring in the course of malarious and other fevers, or of pneumonia, or having a nervous origin, are due to hepatic congestion, and that then the treatment must be modified in conformity with the nature of the primary disease or cause.

3. In the jaundice arising from deficient oxygenation, the chief treatment must be the removal of the cause. In the jaundice of infants, which is independent of obstruction of the bile-duct, the first thing to be done is to put the child in a wholesome atmosphere and to avoid exposing it to cold. The jaundice will then often disappear spontaneously without further treatment; but if it persist, a small dose of hydrargyrum cum creta, followed by castor oil, will often hasten its disappearance.

4. Lastly, in those terrible cases of jaundice associated with cerebral symptoms and the typhoid state, whether occurring in the course of malarious or infectious fevers, or in pyæmia, pneumonia, or the acute yellow atrophy of the liver, or having a purely nervous origin, it is not often that treatment is of much avail in averting a fatal result, but the measures most likely to do good are those which I have already recommended when speaking of the treatment of acute yellow atrophy (see p. 267). Blisters[1] and sinapisms to the nape and scalp, sinapisms to the feet, and remedies calculated to promote elimination by the skin, kidneys, or bowels, are sometimes of service. At the same time it will be necessary to support the patient's strength by diffusible stimulants and small quantities of alcohol.

The following cases illustrate the remarks now made on the subject of jaundice independent of any obstruction of the bile-duct. Most of them occurred in my practice at the London Fever Hospital, where illustrations of the typhoid state, not only in the various specific fevers, but in many other diseases, are probably more numerous than in all the other hospitals of the metropolis put together.

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[1] It is well to avoid cantharides for blistering purposes in these cases, when the urine contains albumen; but a blister can be readily produced, even on the scalp, by applying to the skin for three or four minutes a piece of lint moistened with strong liquor ammoniæ, and covered with oiled silk. I have repeatedly produced a blister in this way with the best effect in cases of typhus complicated with albuminuria and cerebral symptoms.

The first three cases are examples of jaundice occurring in the course of typhus fever—an event, as I have told you, of extreme rarity. In two of the cases leucin and tyrosin were found in the urine: in the remaining cases they were not looked for.

CASE CXXXIII.—*Typhus complicated with Jaundice—Death by Coma—Leucin and Tyrosin, but scarcely any Urea, in Urine—Leucin and Tyrosin in Liver and Kidneys.*

Robert R——, aged 33, admitted into London Fever Hospital August 26, 1862.

On admission, was too confused to given any account of himself; pulse 120, feeble; tongue dry and brown along centre; skin warm and dry, with distinct typhus-rash, and a general yellowish tint. Was ordered beef-tea, milk, brandy (6 oz.), and a mixture containing sulphuric acid, sulphuric ether, and quinine.

Patient became weaker and more unconscious. On 28th, decided jaundice of entire skin and of conjunctivæ; brandy was increased to 8 oz.

August 29.—Pulse 120 and feeble; scarcely conscious, and inclined to be drowsy; pupils contracted. Decided jaundice of skin and conjunctivæ, and at same time a well-marked petechial typhus-rash on chest and abdomen. Involuntary evacuations; tongue brown; motions light-coloured, but contain bile; no tenderness in hepatic region. Urine of a bilious colour, but does not yield reaction of bile-acids; clear, acid, throws down no deposit, and contains no albumen; specific gravity 1017. Six oz. of urine were evaporated, and residuum was found to contain abundance of globular masses of leucin and needle-shaped crystals of tyrosin, and also crystals of triple phosphate. When nitric acid was added to a drop of urine, after concentration to one-twelfth of its volume, only a few small crystals of nitrate of urea could be discovered with the microscope. A blister was applied to the scalp; but patient died comatose, at 3 P.M on Aug. 30.

Autopsy.—Deep jaundiced tint of entire surface. Heart and lungs healthy; blood fluid and dark. Spleen, 7 oz., very soft. Gall-bladder contained bile, which on squeezing flowed readily into duodenum. Liver, 62 oz., rather pale and very friable, but lobules distinct; hepatic tissue contained numerous globular crystalline masses of leucin and tyrosin; secreting cells loaded with oil and bile-pigment. Kidneys enlarged, each weighing upwards of 7 oz.; surfaces smooth; cortex hypertrophied and containing crystalline bodies, similar to those found in liver; uriniferous tubes gorged with epithelium. Intestines healthy, and their contents well coloured with bile.

CASE CXXXIV.—*Typhus Fever complicated with Jaundice.*

Henry B——, aged 42, admitted into London Fever Hospital Sept. 24, 1862. He was in a state of delirium and stupor and quite unable to give any account of himself, but his body was covered with a petechial typhus-eruption, tongue was dry and brown, and pulse 120 and feeble. There was also well-marked general jaundice of skin and conjunctivæ, with bile-pigment and albumen in urine. Abdomen distended and tympanitic, but no tenderness nor enlargement of liver. Bowels rather loose and motions dark. Treatment consisted in nitro-muriatic acid, nitrous ether and taraxacum, beef-tea, milk, wine, and subsequently brandy.

The jaundice increased, and, although pulse fell to 84, patient became weaker, urine had to be drawn off by catheter, and death took place on Sept. 27.

Autopsy.—Intestines contained bile, and there was no obstruction of bile-duct. Liver pale and slightly fatty; spleen large and soft. Uriniferous tubes of kidneys gorged with granular epithelium.

CASE CXXXV.—*Typhus Fever—Double Pleuro-pneumonia—Jaundice—Tyrosin in Urine.*

James P——, aged 47, was admitted into London Fever Hospital on Feb. 23, 1864, with usual symptoms of a severe attack of typhus fever, duration of which was doubtful. On admission, pulse 128 and feeble; distinct typhus-eruption; tongue dry and brown; bowels confined; mind confused and occasional delirium; signs of congestion at bases of both lungs. He was ordered mineral acids with ether, 6 oz. of brandy; milk, beef-tea, and an egg; and mustard and linseed poultices to back of chest.

On Feb. 26 patient had tremors and subsultus and was lower; a decided yellowness of skin and conjunctivæ was noticed, but there was no enlargement nor tenderness of liver. Respirations easy, and lungs resonant on percussion. Urine threw down a copious deposit of lithates both on Feb. 26 and 27; and on latter day respirations 40; dulness with tubular breathing over lower third of both lungs; and jaundice was more decided. Patient was ordered a mixture of ammonia, ether and senega, and brandy was increased to 10 oz..

On Feb. 28, pulse 128 and respirations 40; dulness of lungs had extended. Jaundice well marked, but plenty of bile in motions. Urine was coloured with bile and gave a distinct reaction of bile-pigment with nitric acid, but contained no bile-acids (by Harley's test); specific gravity 1018; a moderate amount of albumen (about $\frac{1}{16}$). On evaporating it down to a syrup, there were found numerous crystals of triple phosphate and yellowish-brown crystalline globules of tyrosin.

On Feb. 29 the pulse 140; respirations 60; rash fading, but body covered with profuse perspiration. He died at 8 P.M., on what was probably about fourteenth day of illness.

Autopsy.—Jaundiced tint of skin and conjunctivæ well marked. Bile in duodenum; bile-ducts quite pervious. Liver not at all congested, but pale, soft, and very friable; lobules distinct, but secreting cells in their pale rims were loaded with oil. Spleen large and diffluent. Kidneys appeared normal, except that cortices were pale and epithelium cells opaque and granular. No leucin nor tyrosin could be detected in hepatic tissue, nor in kidneys. Lower lobes and lower part of upper lobes of both lungs were in a state of grey granular consolidation, and pleural surfaces of inflamed lungs were coated with a thin film of recent lymph.

In the last case the double pneumonia no doubt contributed to the development of the jaundice. In the one which follows the jaundice was a sequel of typhus, and coexisted with phlegmasia dolens and fatty degeneration of the liver, kidneys, and heart.

CASE CXXXVI.—*Typhus Fever, followed by Phlegmasia Dolens, Jaundice, and Death.*

Rosetta J——, aged 42, admitted into London Fever Hospital Feb. 24, 1857. This patient had been ill for about eight or nine days before admission; and after she came under observation in hospital most prominent symptoms were: pulse 120; extreme prostration; great restlessness and much low muttering delirium; involuntary stools and urine; well-marked typhus-rash; dry, brown tongue, and constipated bowels. Treatment consisted in wine, carbonate of ammonia, and castor oil to keep bowels open.

About five or six days after admission an improvement took place in symptoms; and, by March 6, she had regained strength to a considerable degree, appetite was good, and pulse 80.

On March 9, which was about 23rd day from first commencement of fever and the 6th of convalescence, patient felt ill again. Pulse 120 and small. Complained much of shooting pains in left leg. Skin hot and dry. Some flushing of face. Tongue moist and very red. The next day considerable swelling and some tenderness of left leg and thigh, but no hardness in course of femoral vein. Heart's action heaving and tumultuous, but no bellows-murmur. Breathing short and rapid; no cerebral symptoms. Blister over heart. Wine ℥vi. Saline efferv. mixture with tinct. hyoscy. ʒss every four hours. Left leg to be fomented and kept elevated.

No improvement took place, but at 4 A.M. of March 12 (fourth day from first complaint of pains in leg) patient felt cold and chilly.

There was a great increase of prostration and pulse was imperceptible, although heart's action continued tumultuous as before. Breathing very rapid. Mental faculties unimpaired. Skin and conjunctivæ of a marked yellow tint, and face livid. Profuse sweating. No tenderness over liver, nor obvious increase of hepatic dulness. Brandy and wine were freely administered, but patient gradually sank and died towards evening.

Autopsy.—Cadaveric rigidity well-marked. Distinct yellow tinge of skin on scalp, neck, and trunk. Thick layer of subcutaneous fat over chest and abdomen. Copious sudamina over chest. Left leg swollen. Left ankle 8¾ in. in circumference; right, 8¼; left calf, 13 in.; right, 11½; left thigh, 17 in.; right, 14½. Cerebral membranes moderately congested and separated readily from brain. Sub-arachnoid fluid and fluid in ventricles of a decided yellow tint. Substance of brain tolerably firm; red points numerous. Half an ounce of yellow serum in pericardial sac. Heart 8¾ oz.; valves normal; left cavities empty, and right almost empty. Walls of right ventricle very thin, and at apex composed almost entirely of fat. Substance of heart pale and soft, and on microscopic examination transverse striation indistinct and fibres presented a granular aspect. Left femoral and iliac veins healthy and contained no adherent clot. Each of lungs weighed 25 oz.; left adherent throughout and very emphysematous; lower lobes of both lungs much congested; no consolidation. Stomach and intestines healthy. Liver 52 oz.; capsule separated readily; substance of organ pale and very soft and friable, so that it broke down on removal; all trace of lobules had disappeared, cut surface presenting a marrow-coloured pulpy appearance. On microscopic examination, many of secreting cells could be seen loaded with oil; others appeared to be breaking up and disintegrating, and much free oil and granular matter. A small quantity of thick bile in gall-bladder; bile-ducts quite pervious. Spleen 13 oz., soft and pulpy. Kidneys enlarged; left 7½ oz., right 7 oz.; capsules separated readily; outer surface smooth; substance pale and flabby; cortical substance pale and granular and rather increased in amount; uriniferous tubes gorged with oily epithelium.

The following are three of the four instances of enteric fever complicated with jaundice which I have met with. In the first the jaundice occurred during a relapse of the fever, and was probably catarrhal; in the second it came on during the acme of the fever and persisted during convalescence; in the third it coexisted with thrombosis of the femoral vein and albuminuria.

Case CXXXVII.—*Enteric Fever followed by a Relapse, with Jaundice.*

Mary A. C——, aged 43, was admitted into London Fever Hospital suffering from enteric fever on Feb. 9, 1863. She had a dry, red, fissured tongue, with diarrhœa and rose-spots. She had been ill for nine days before admission, and on Feb. 25 she began to convalesce. She improved daily until March 5, when febrile symptoms and diarrhœa returned, and on March 8 fresh rose-spots were observed. On March 11 conjunctivæ and skin first became yellow, and on March 14 there was deep jaundice of whole surface. Urine was dark green, deposited much lithates, and contained much bile-pigment, but no albumen, leucin, nor tyrosin. The bowels, from appearance of jaundice, were rather confined, and motions clay-coloured. Hepatic dulness measured 4 in. in right mammary line; no tenderness below right ribs. Tongue dry; great prostration; but no delirium. Patient was treated with nitro-muriatic acid and gentian, and linseed-poultices over abdomen. On March 16 jaundice began to subside, and by 21st it had almost disappeared and patient was again convalescent.

The following case was communicated to me in a letter from the patient, who is a Fellow of the Royal College of Physicians.

Case CXXXVIII.—*Enteric Fever complicated with Jaundice.*

One remark struck me in your book on Fevers. You refer to the extreme rarity of the complication of jaundice with typhoid. I myself was the subject of this affection in a very intense degree. This was in Paris in 1842. The jaundice came on suddenly about the acme of the fever. When the event was told to Rostan, who was seeing me, he said it was a 'complication bien facheuse,' and he did not expect that I would recover. This deep jaundice persisted for some time, even during convalescence, so that when I used to crawl into the Luxembourg Gardens I was known amongst the frequenters as the 'Monsieur Jaune.'

Case CXXXIX.—*Enteric Fever complicated with Jaundice and Thrombosis of Femoral Vein.*

On Dec. 12, 1863, I was requested by Mr. Edward Newton to see Mr. W——, a gentleman about 54 years of age, who on Sept. 30 had been taken ill with enteric fever, which presented the usual symptoms of diarrhœa, rose-spots, &c. till fourteenth day, when he became jaundiced and albumen appeared in urine. During convalescence he got thrombosis of left femoral vein, with considerable tenderness along the vein; but after ten days this subsided and the albumen disappeared from urine. About a week before I saw him, he had driven out to country, got out of the carriage and walked for five minutes. Within a few hours pain and swelling in leg returned, and when I saw him on Dec. 12 there was considerable pitting of left leg, but only slight tenderness along vein. Urine was turbid; sp. gr. 1013; it contained one-eighth of albumen and granular epithelial casts, but only a faint trace of bile-pigment, and no leucin nor tyrosin. Impulse of heart barely perceptible, and first sound short and abrupt like second.

The patient was treated with iron, quinine, a generous diet, and wine. At first he rallied somewhat, and on Jan. 9 swelling had almost left legs, there was no jaundice, and only a trace of albumen in urine; but soon after this he became weaker, and he died at St. Leonards in March 1864. There was no post-mortem examination.

In the four cases which follow, jaundice appeared in the course of scarlet fever. In the first three, two of which were fatal, the symptoms indicated serious blood-changes; in the fourth the jaundice was probably the result of simple congestion.

CASE CXL.—*Scarlatina—Jaundice—Death by Coma.*

Samuel W——, aged 27, was admitted into London Fever Hospital on March 6, 1863, having been ill with fever and sore throat for four or five days. On admission, pulse 120, weak. Copious, bright, punctated, scarlet eruption. Skin, especially of face, and conjunctivæ distinctly jaundiced. Tongue very red at edges, dry and brown along centre; throat sore; tonsils red and enlarged, not ulcerated; occasional hiccough; had vomiting and some purging before admission; no nasal discharge; mind clear. Ordered: carbonate of ammonia and chlorate of potash, 5 gr. of each every four hours. Wine 6 oz.; beef-tea and milk.

During night patient became quite unconscious and face was dusky and livid, and on following morning at 8.30 A.M. he died.

Autopsy.—Skin and white tissues deeply jaundiced. Bile-ducts patent. Liver presented a nutmeg appearance on section, margins of lobules being pale and their central vessels containing blood; oily matter in secreting cells much increased. Kidneys large, right weighing 6¾ oz. and left 8¼ oz.; capsules non-adherent and surfaces smooth; cortices hypertrophied, five or six lines in thickness, dark-red and dripping with blood; uriniferous tubes gorged with finely granular epithelium. Urine from bladder had a specific gravity of 1015, and contained a considerable amount of albumen and bile-pigment.

CASE CXLI.—*Scarlatina—Jaundice—Sudden Death.*

Alfred C——, aged 19, was admitted into London Fever Hospital on Dec. 4, 1862. His illness had commenced four days before with pains in limbs and sore throat, followed by a scarlet rash which was now well out. Pulse 130; tongue moist, with white coat and red edges; bowels open; tonsils swollen and red, not ulcerated; no nasal discharge. Ordered, quinine (2 gr. every four hours), milk and beef-tea.

Dec. 5 (6th day).—Pulse 136 and feeble. Had a restless night, wandering occasionally, but slept at intervals. Swallows liquids well. Rash still out. Bowels open. Ordered 4 oz. of wine.

Dec. 6 (7th day).—Was restless during night, but slept at intervals. This morning nurse observed lips to be slightly livid and face and conjunctivæ yellow, but otherwise man seemed no worse. He asked nurse to put his tea down as it was too hot. Ten minutes later nurse saw him again and found him unconscious, breathing quickly, and he died in five minutes, at 8.30 A.M.

Autopsy.—All white tissues were tinged yellow, and lungs were moderately congested posteriorly. Liver and bile-ducts presented nothing abnormal, except that former was pale and slightly fatty. Kidneys considerably congested; uriniferous tubes gorged with granular epithelium; urine in bladder contained a small quantity of albumen.

CASE CXLII.—*Scarlatina—Jaundice—Recovery.*

Emily S——, aged 18, was admitted into Fever Hospital April 5, 1864, having been ill one day with fever and sore throat. On admission, pulse 120; skin very hot; punctated scarlet rash of good colour and moderate intensity. Tongue red, with white fur, and dry along centre; fauces red; tonsils large; no ulcer. No enlargement of glands in neck. Ordered a mixture with chlorate of potash and free chlorine, beef-tea, &c.

April 6 (3rd day).—Pulse 120, feeble. Bowels open three or four times since last night. Ordered four ounces of wine and an egg.

April 7.—During early part of last night was very restless and wandered, but slept fairly after an opiate draught. To-day, pulse 130; tongue dry; no ulcer of tonsils: answers correctly; intense scarlet rash on arms.

April 9 (6th day).—Pulse 120, very feeble; rash fading; entire skin and conjunctivæ present a light jaundiced tint; no tenderness over liver; bowels open twice; motions pale yellow; tongue dry; desquamation commencing on face; slept badly, and mind wanders at times. Urine contains bile-pigment, but no albumen. Ordered 6 oz. of brandy.

April 11.—Pulse 96; skin cooler; desquamation general; jaundice as before; three light-yellow stools; mind clearer; appetite returning, and girl feels better. Ordered quinine and custard pudding.

April 13.—Copious desquamation. Jaundice fading.

April 17 (14th day).—General health improving. To-day, for first time, skin is free from yellow tint. Bowels still slightly relaxed.

Was discharged well on April 29.

CASE CXLIII.—*Scarlatina—Jaundice—Recovery.*

Frederick C——, aged 27, was admitted into London Fever Hosp. Dec. 17, 1861. Wife and child in hospital with well marked scarlatina. Was taken ill day before with sore throat, rigors, and headache; and on admission, pulse 114; faint scarlet rash; tongue moist and furred, red at tip and edges; swallows with pain; tonsils large and red, but not ulcerated; bowels open. Was ordered an acid mixture, low diet, and beef-tea.

Dec. 19 (4th day).—Face somewht yellow; some tenderness over liver. Ordered two aperient pills.

Dec. 20.—Jaundice more decided; conjunctivæ yellow; tongue moist, clean and red; throat less sore; bowels open three times; plenty of bile in motions; rash gone; pulse 84; and feels better.

Dec. 21 (6th day).—Jaundice more intense; urine contains much bile-pigment, but no albumen; slight tenderness on pressure over liver; bowels confined. Ordered a mixture containing nitrate of potash and sulphate of magnesia.

Dec. 23.—Pulse 66; less jaundice; no pain in hepatic region; bowels open several times.

From this date patient continued to improve until discharge on Jan. 26, 1862. The jaundice disappeared in a few days. Desquamation was slight.

I have already brought under your notice several cases where jaundice resulted from pyæmia and where multiple abscesses were found in the liver (Cases LXVII. to LXXIII. p. 169). In the following case the jaundice was also due to pyæmia, but there were no purulent deposits in the liver, and at the same time the bile-ducts were perfectly patent.

CASE CXLIV.—*Acute Necrosis of Cervical Vertebræ—Pyæmia—Jaundice.*

Elizabeth A——, aged 24, admitted into London Fever Hosp. on Feb. 17, 1868. She had been on the streets prior to her marriage and had scars of buboes in groins. Twelve days before admission she had been suddenly seized with acute pain in back of neck, which had never left her, had prevented all motion of her head, and was accompanied by vomiting. She fancied that she had injured her neck while wrestling a week before her seizure, but her friends attached no importance to this as a cause of her illness.

On admission, patient was suffering from symptoms of general fever, without any indication of local disease excepting great pain and tenderness in back of neck and in both shoulders, increased by movement, but without any obvious swelling or induration. Pulse 108; tongue moist and red. Mind clear. No eruption on skin, which was hot, with occasional perspirations; no rigors. A blister was applied to back of neck, and a mixture containing iodide of potassium and carbonate of ammonia (āā gr. iv) and extract of belladonna (gr. $\frac{1}{3}$) was prescribed.

A few days after admission an obscure swelling could be felt down neck on either side of cervical vertebræ; there was a circumscribed pink flush on left cheek; tongue dry and brown down centre; and patient complained of great burning and dryness of throat. Still occasional vomiting, and pulse had risen to 126. On Feb. 28 patient had several severe rigors; she had been slightly delirious in night; diarrhœa with copious watery motions; a distinct pericardial friction over heart, but no albumen in urine. On March 2 there was slight, but distinct, jaundice of skin and conjunctivæ and bile-pigment in urine, but there was no enlargement nor tenderness of liver, and motions, which were still watery, contained plenty of bile; pulse 144; no return of rigors and no perspirations. A fluctuating swelling, size of a small orange, was discovered a little behind left ear, which on March 4 was opened and discharged a quantity of thick pus. Patient was extremely prostrate, scarcely conscious, and very restless. Jaundice increased in intensity until death on March 5.

Autopsy.—Laminæ of all cervical vertebræ excepting first and two last bare and bathed in pus; also pus in spinal canal external to theca and in left lateral sinus. Left lung contained five or six small patches of lobular pneumonia passing into pus. Liver seemed healthy, except that secreting cells contained too much oil; it was not congested, and bile-ducts were perfectly patent. No ulceration of the intestines.

Case CXLV. is an example of jaundice occurring in the course of acute pleuro-pneumonia.

Case CXLV.—*Acute Pleuro-pneumonia complicated with Jaundice.*

On May 23, 1867, I was requested by Dr. W. H. Cook, of Hampstead, to see a clergyman, between 50 and 60 years of age, of spare build and temperate habits, who had been long subject to spasmodic asthma, and who, five days before, had been seized with severe pain in right hypochondrium and febrile symptoms, followed by slight jaundice, cough, and high-coloured urine. On examination we discovered all the physical signs of pleuro-pneumonia of lower half of right lung—dulness, tubular breathing, fine crepitation, friction, and increased vocal resonance. There were also cough with considerable dyspnœa and tenacious rusty sputa, and acute pain in right side, greatly aggravated by coughing or by taking a long inspiration. Pulse 120; respirations 36. Along with these signs of pulmonary disease there was slight but decided jaundice of skin and conjunctivæ, and urine gave evidence of presence of bile-pigment. Liver projected about half an inch below ribs in right mammary line; slight tenderness over it on firm pressure; no deficiency of bile in motions. Tongue dry and brown. Great despondency as to result of his illness. Patient was treated with carbonate of ammonia, nitrous ether, stimulants, opiates, and other anodynes to relieve pain and procure sleep, and mustard and linseed poultices to side.

At first there was a marked improvement in both physical signs and general symptoms, but early in June pleurisy and then pneumonia made their appearance in left lung, and patient continued to sink until death on June 14. To the last there was a slight icteroid tinge of skin and conjunctivæ, but motions always contained plenty of bile. There was no post-mortem examination.

Case CXLVI. appears to be a well-marked instance of jaundice from congestion of the liver. The fact of the jaundice following an attack of acute rheumatism, from Dr. Graves's observations (see *antea*, p. 318), led us to look for urticaria, but no such eruption appeared.

CASE CXLVI.—*Jaundice from Congestion of Liver.*

Jane G——, aged 30, was admitted into Middlesex Hospital on April 2, 1868. You will remember her as a patient in Seymour Ward from January 28 to March 9, suffering from acute rheumatism and pericarditis. After leaving hospital she had complained of weakness and rheumatic pains, but she had been gradually getting better until March 26, when she was attacked with pain in region of liver, back and front, which on March 28 became much worse and was followed on next day by nausea, vomiting, and great flatulence, and on 30th by jaundice. Vomiting only lasted for a day, but jaundice and pain continued to increase.

On admission well-marked jaundice of entire skin and conjunctivæ, and patient complained greatly of pain in epigastrium and right hypochondrium, stretching up to right shoulder and down back. Pain was greatly increased by pressure below right ribs and also by lying on left side, which patient said always took away her breath. Area of hepatic dulness was increased, in r. m. l. measuring $5\frac{1}{4}$ in. and extending quite an inch beyond margin of ribs. Urine was acid and dark, and contained a considerable amount of bile-pigment. Tongue furred; no appetite; bowels had been very freely moved by a dose of compound jalap powder taken on day before admission, and motions had contained plenty of bile. Pulse 96 and temperature 100°.

The whole hepatic region was dry-cupped and afterwards covered by linseed-poultices; a draught of sulphate of magnesia and senna was ordered, with a diuretic mixture containing bitartrate of potash and nitrous ether. Diet was restricted to milk and beef-tea.

On following morning catamenia appeared for first time since patient's confinement four months before; frequent bilious motions; pain and tenderness in region of liver greatly relieved.

On April 8 patient was free from pain and jaundice scarcely perceptible. A mixture with nitric acid and gentian was now prescribed and fish diet.

On April 13 jaundice quite gone; hepatic dulness in r. m. l. only 4 in.; no tenderness below right ribs. On following day patient was discharged.

The last case which I shall mention is remarkable not only for the persistence, but for the hereditary character of the jaundice. Although there may be doubts as to whether the yellowness of the children of the third generation was true jaundice, there can be no question as to its reality in the case of the mother and her two sons. It must be admitted that the pathology of the jaundice in these cases is obscure. It is clear that there is little or no obstruction of the bile-duct; and the most probable view is that the liver either secretes an excess of bile, or is in a permanent state of congestion (see p. 412). But whatever be its pathology, its intimate relation to gout is interesting in relation to the view which I shall hereafter submit to your notice, that gout has its origin in functional derangement of the liver.

CASE CXLVII.—*Hereditary Jaundice and Gout.*

On Feb. 18, 1875, I had, through the kindness of Dr. Moxon, an opportunity of seeing the following case.

Robert J——, aged 30, says he was born yellow and that he has been jaundiced as long as he can remember. He had never been robust, but from age of 17 to 27 he had been in habit of walking twenty miles a day, exposed to all kinds of weather and eating all sorts of food. At 21 he had been laid up for six weeks with a febrile attack; and at 27 he had another illness, after which he had led a more sedentary life, but had continued to drink about three pints of beer daily. From 27 up to the date of his visit to me he had suffered much from gout in fingers and toes, which had always been relieved by iodide of potassium. Urine had deposited a copious sediment as long as he could remember. Four months before seeing me he had left off beer and all stimulants, and during that time he had become much fairer than ever before.

I made following notes: Body fairly nourished, and is leading an active life. Has decided yellowness of skin and conjunctivæ. Urine contains abundance of lithates, and presents distinct reaction of bile-pigment. Liver slightly enlarged, measuring 4¾ in. in r. m. l., not tender. Appetite good; occasional flatulence; bowels sluggish; motions fairly coloured with bile. Always has giddiness on looking up, and formerly had it whenever he got up or lay down.

On Oct. 9, 1876, Dr. Moxon wrote to me as follows: R. J. is still a teetotaller; but urine contains bile-pigment as before and jaundice persists.

R. J. has a brother, two years and eight months older than himself, who has also been deeply jaundiced all his life and in Feb. 1875 was laid up with gout. In October 1876 I heard from Dr. Moxon that this brother continued to drink beer and was still very much jaundiced.

The mother of these two brothers died at age of 54. She had been in habit of drinking malt liquor freely, and for fourteen years before death she had been a great sufferer from gout, her feet and every one of her finger-joints being greatly deformed from it. During same period she had been continuously jaundiced, the 'whites' of her eyes being deep yellow, and she had had frequent bilious attacks. She had borne seven children, but five had died soon after birth or in early infancy. An only brother had died of consumption; but her father had suffered greatly from gout and liver-complaints.

Father of R. J. is still (Oct. 1876), alive and healthy; never had gout nor jaundice.

I learn from Dr. Moxon also that both R. J. and his brother have had several children, all of whom 'became deeply jaundiced two days after birth, the colour being—eyes, body, and whole frame—as deep as possible, but disappearing after about a month.'

Before concluding this lecture I must say a few words on the diagnosis of the causes of jaundice, and more especially on the detection of bile-acids in the urine as an aid to diagnosis. In 1858 Kuhne announced that 'in jaundice resulting from closure of the ductus communis choledochus, the urine always contains biliary acid as well as bile-pigment;' but that under ordinary circumstances, when the ducts are free, 'the bile-acids for the most part pass off by the fæces and are not reabsorbed from the intestine.'[1] Dr. G. Harley, in his Essay on Jaundice, published in 1863, adopting the view that bile-acids are formed by the liver, while bile-pigment is pre-formed in the blood (see *antea*, p. 326), maintained that the detection of bile-acids in the urine was a means of distinguishing jaundice due to obstruction from that due to suppressed secretion. 'In jaundice from suppression,' Dr. Harley remarks, 'the liver does not secrete bile; consequently no bile-acids being formed, none can enter the circulation, and they are therefore not to be detected in the urine;' whereas in jaundice from obstruction 'bile is secreted and absorbed into the blood, and the bile-acids not being all transformed in the circulation are eliminated by the kidneys and appear in the urine' (p. 60). Accordingly, it is argued that the presence or absence of bile-acids in the urine, in any case of jaundice, ought to decide whether this be due to obstruction or suppression; and the readiest mode by which the bile-acids may be detected is said to be the following application of Pettenkofer's test: 'To a couple of drachms of the suspected urine add a small fragment of loaf-sugar, and afterwards pour slowly into the test-tube about a drachm of strong sulphuric acid. This should be done so as not to mix the two liquids. If biliary acids be present, there will be observed at the line of contact of the acid and urine—after standing for a few minutes—a deep purple hue. This result may be taken as a sure indication that the jaundice is due to obstructed bile-ducts.' On the other hand, 'a brown instead of a purple tint' is said to be equally indicative of suppression (p. 61).

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[1] Virchow's Archiv, Sept. 1858; and Translator's Preface to the English edition of Frerichs on the Liver, vol. i. pp. 14, 15, June 1860.

If the views here announced should be confirmed, the pathology of jaundice would be greatly simplified and physiological chemistry would have contributed an important aid to diagnosis. But after having bestowed considerable attention on the matter, it appears to me that both the theory and the practice based on it are open to objection.

1. For reasons already given (p. 327), the theory that jaundice independent of obstruction of the bile-ducts is due to *suppressed secretion* of bile is itself a very improbable one.

2. Supposing the theory to be correct, bile-acids might be present in the urine unless the liver were *entirely* destroyed or the secretion *wholly* suppressed—events admitted to be of rare occurrence. In cases also of obstruction of long standing, the secreting tissue of the liver, as Dr. Harley admits, may be destroyed, and accordingly bile-acids might be absent from the urine, although the jaundice resulted in the first instance from obstruction. In point of fact, Golowin and other recent observers have found that in cases of jaundice from obstruction the bile-acids ultimately disappear altogether. It follows that, however correct the theory, its practical application may mislead the physician in diagnosing the cause of jaundice.

3. The clinical evidence adduced in corroboration of the view that jaundice from obstruction is to be recognised by the presence of bile-acids in the urine is as yet insufficient. Neither Scherer,[1] a chemist of note, nor Frerichs has ever found bile-acids in the urine in any form of jaundice; and although minute quantities may be detected by delicate tests such as that of Hoppe, after separating the urinary pigments, the necessity of which Kuhne admits, these methods are scarcely applicable to the ordinary purposes of diagnosis. Of the five cases of jaundice recorded by Dr. Harley,[2] in which the urine was tested for bile-acids, one was a case of jaundice from obstruction of the common duct where abundant bile-acids were discovered on one occasion, but not a trace of them ten days afterwards; although seven weeks later, shortly before death, they reappeared in small quantity (p. 74). Another was a case of acute yellow atrophy of the liver, in which a decided reaction of bile-acids in the urine was obtained. This discovery might have been thought fatal to the diagnostic significance attributed to bile-acids in the urine, inasmuch as in acute yellow atrophy the bile-ducts are perfectly patent and the disease has usually been regarded as a typical illustration of jaundice from suppression; but Dr. Harley is of opinion that the presence of bile-acids in this case proves that in acute atrophy the suppression is complicated with reabsorption of bile (pp. 33, 38).

4. Since the promulgation of Dr. Harley's views, I have tested the urine in a large number of cases of jaundice, and have serious doubts as to Dr. Harley's modification of Pettenkofer's test, in which there is no provision for separating in the first place the urinary pigments, being a reliable indication of the presence of bile-acids in the urine.[3] You will remember that on one occasion I applied the test to the urine of six patients under my care in the Middlesex Hospital. In three of the six cases a dark purple colour was developed at the line of junction of the sulphuric acid and the urine. One of the three cases was an example of jaundice from impacted gall-stone: in the other two cases, there was neither jaundice, nor any symptom of disease of the liver, and yet when the three

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[1] Chemical Gazette, vol. ii. 1845, p. 208.

[2] Op. cit. pp. 27, 38, 74, 94, 111.

[3] Dr. Hilton Fagge has arrived at a similar conclusion. Guy's Hosp. Rep. 1875. vol. xx.

test-tubes were placed side by side, it was impossible to distinguish the colour in the first from that in the remaining two. Other observers, I believe, have arrived at similar results. I find also that Neubauer, in his excellent monograph on the Urine,[1] states that certain of the pigments of the urine produce a dark purple-violet colour exactly resembling that from bile, when a large quantity of strong sulphuric acid is added to the urine. To separate the urinary pigments before testing for the bile-acids would obviously be too tedious and difficult a process for the ordinary purposes of diagnosis, even supposing that the presence or absence of the bile-acids threw more light on the cause of jaundice than it probably does.

Although from these considerations I regret that I cannot recommend the test to which I have been adverting, as furnishing reliable information as to the cause of jaundice, the subject is one which you will do well to investigate for yourselves. Meanwhile, in forming a diagnosis you will sometimes be assisted by bearing in mind the remarks with which I bring this lecture to a close

1. The chief indication of obstruction of the common bile-duct is furnished by the stools. When there is no obstruction of the duct, the stools contain bile; but when the duct is obstructed, no bile enters the bowel and the stools are clay-coloured. Several sources of fallacy must be remembered. First, the jaundice usually persists for a short time after the removal of the obstruction, and thus, as happens not unfrequently in the case of gall-stones, bilious motions may coexist with jaundice which has resulted from obstructed bile-ducts. Secondly, if the motions be thin or watery, they may appear to contain bile from the admixture of jaundiced urine. Thirdly, in rare cases the bile-duct is only partially occluded and sufficient bile passes to colour the fæces. And fourthly, some cases of jaundice have a complex origin, there being both occlusion of the duct and a morbid state of blood.

2. A tumour corresponding to the region of the gall-bladder will favour the view that the jaundice is due to obstruction of the bile-duct (p. 161).

3. Jaundice which persists, and is yet slight, is most probably independent of obstruction of the bile-duct. Persistent jaundice from obstruction speedily becomes intense;

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[1] A guide to the qualitative and quantitative Analysis of the Urine, by Dr. C. Neubauer and Dr. J. Vogel, Syd. Soc. Transl. p. 48.

but in reference to this you must remember, what I have so often insisted upon, that even when there is irremovable obstruction of the bile-duct, the intensity of the jaundice will vary from time to time according to the amount of bile secreted by the liver and the activity of the kidneys, and that in the advanced stage the jaundice may permanently fade in consequence of the destruction of the glandular tissue and the small quantity of bile which is secreted.

4. Jaundice appearing suddenly in a person whose previous health has been good is most probably the result of obstruction of the duct by a gall-stone, or it has a nervous origin. In the former case it will be preceded or accompanied by biliary colic and vomiting, and the stools will be clay-coloured: in the latter there will be a history of concussion or of some severe mental emotion, the motions will contain bile, and the jaundice will be often accompanied by delirium and other cerebrals ymptoms.

5. Jaundice coming on very slowly, but ultimately becoming intense, with complete disappearance of bile from the motions, is most probably the result of pressure on the bile-duct from without, or of the growth of some tumour in the interior of the duct (pp. 352–3).

6. Several attacks of temporary jaundice with distinct intermissions point to gall-stones, if the patient be of adult or advanced life (p. 340); in early life, to catarrh of the duodenum or bile-ducts (p. 346).

7. Pain is present in some cases of jaundice, absent in others. There may be little or no pain in cases where the cause is a duodenal ulcer, a simple stricture of the duct, enlarged glands in the fissure of the liver, or the poison of some specific fever. It is well also to remember that in very rare cases a gall-stone has been known to obstruct the common duct and cause permanent jaundice, without ever having excited attacks of biliary colic. A pain coming on in severe paroxysms, and then subsiding, may result from: *a*, gall-stones (see p. 337); *b*, hydatids (p. 344); *c*, a duodenal ulcer (p. 349); and, *d*, an aneurism of the hepatic artery (p. 357). Jaundice immediately preceded by severe paroxsyms of pain is most probably due to gall-stones; jaundice followed by severe paroxysmal pain is more probably the result of cancer. Pain, more or less constant, with tenderness on pressure below the right ribs, will indicate that the jaundice depends on: *a*, con-

gestion of the liver (p. 132); *b*, interstitial hepatitis (p. 139); *c*, catarrh of the bile-ducts (p. 152); *d*, pyæmia with purulent deposits in the liver (p. 165); *e*, cancer of the liver (p. 210), *f*, acute atrophy of the liver (p. 261). Lastly, in reference to the concurrence of pain and jaundice you must remember that cancer of the gall-bladder is not an uncommon sequel of gall-stones.

8. Jaundice concurring with great enlargement of the liver is most probably due to cancer of the liver (p. 208); but it may also arise from waxy liver, when the bile-duct is compressed by enlarged glands in the portal fissure (p. 33), from multiple abscesses of the liver (p. 167), or from interstitial hepatitis (p. 140).

9. The diagnosis of the cause of jaundice is often materially assisted by the coexistence with it of ascites. When these two conditions coexist, you will usually find that there is either cancer or cirrhosis. When permanent jaundice with complete absence of bile from the motions, and ascites without dropsy elsewhere, are present in the same case, you will rarely be wrong in inferring that the obstruction of the gall-duct which causes the jaundice, and the obstruction of the portal vein from which the ascites results, are due to a common cause. That cause cannot be a gall-stone. This will obstruct the bile-duct, but cannot obstruct the flow of blood in the portal vein, so as to produce ascites.[1] The double obstruction is most likely to be caused by pressure from without upon the gall-duct and portal vein, where they lie side by side in the fissure of the liver, by enlarged lymphatic glands, by a tumour in the head of the pancreas, or by cancerous nodules projecting from the surface of the liver itself. It is quite possible, however, for these lesions to cause jaundice without ascites. In the advanced stage of cirrhosis it is also not uncommon for jaundice to coexist with ascites; but then the liver is often small, the jaundice is slight, little more than sallowness, and, what is more important, the colour of the motions proves that bile-pigment is still secreted, and finds its way into the bowel. There is not a complete absence of bile-pigment from the excrement.

10. In a large proportion of cases of jaundice the pulse is unusually slow and the temperature is not increased. When jaundice is accompanied by febrile symptoms, the probable causes are: *a*, inflammation or ulceration of the bile-ducts (p. 153); *b*, some specific fever (p. 395): *c*, pyæmia (pp. 165, 404); tubercle (p. 245); or inflamed hydatid (p. 69).

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[1] But see Lecture XIII., Case CLXVII.

11. Delirium, stupor, and other cerebral symptoms concurring with jaundice suggest: *a*, acute atrophy of the liver (pp. 262, 322); *b*, poisoning by phosphorus (p. 405); *c*, some specific fever or other blood-poison (p. 395); *d*, nervous shock (p. 409); or *e*, pneumonia (p. 411). In all these cases the symptoms are those of an acute illness, the stools contain bile, and the urine often contains leucin and tyrosin and is deficient in urea. Similar symptoms, however, may also supervene in cases of protracted jaundice from obstruction of the bile-duct, in which the stools contain no bile (p. 273).

12. In diagnosing the cause of jaundice it is always important to keep in view the condition of the patient prior to its appearance. In the case of jaundice from gall-stones or nervous shock the patient may have been in excellent health previously. In catarrhal jaundice the attack is preceded for a week or ten days by gastric symptoms with vomiting or diarrhœa (p. 152). Emaciation with loss of appetite, flatulence, and vomiting of food prior to the jaundice ought to suggest cancer of the pancreas, duodenum, or pylorus (pp. 352, 354), and pain two or three hours after a meal with attacks of hæmatemesis or melæna will point to a duodenal ulcer (p. 348). Jaundice occurring in the course of specific fevers or pyæmia will be preceded by the symptoms characteristic of these disorders. Jaundice in the early stage of pregnancy may be due to congestion of the liver from suppression of the catamenia (p. 134); in the more advanced stages it may arise from pressure of the enlarged uterus upon the bile-duct (p. 358), or from acute atrophy (p. 265). Lastly, true jaundice in the new-born child may result from the inhalation of a vitiated atmosphere (p. 411), from plugging of the bile-duct by inspissated bile or gall-stones (p. 342), or from congenital closure or deficiency of the duct (p. 346).

LECTURE XII.

FLUID IN THE PERITONEUM.

ITS SIGNS—THE CONDITIONS WHICH SIMULATE IT, AND HOW TO DISTINGUISH THEM: 1. OVARIAN CYST: 2. HYDATID TUMOUR; 3. RENAL CYST; 4. DISTENDED URINARY BLADDER; 5. PREGNANT UTERUS—CAUSES OF FLUID IN PERITONEUM: I. ACUTE PERITONITIS; II. TUBERCULAR PERITONITIS; III. CHRONIC PERITONITIS; IV. CANCER; V. COLLOID DISEASE; VI. SIMPLE DROPSY—1. FROM DISEASE OF KIDNEYS; 2. FROM DISEASE OF HEART OR LUNGS; 3. FROM PORTAL OBSTRUCTION.

GENTLEMEN,—Pursuing a similar course to that adopted when I was lecturing on enlargements of the liver and jaundice, I purpose to-day laying before you the various causes of fluid in the peritoneum and the means of distinguishing them, more especially in reference to diseases of the liver, of which ascites is so common a symptom.

The signs of fluid in the abdominal cavity are as follows:—

1. There is enlargement or swelling of the abdomen.

2. A dull sound is elicited on percussion over the seat of fluid. It is very common for persons in middle or advanced life to consult a medical man in the belief that they have got dropsy, the swelling being nothing more than an accumulation of gas in the bowels, aided perhaps by an increase of the subcutaneous fat. The nature of the case will be at once revealed by percussion, which will give forth a clear sound over a tympanitic bowel. In rarer cases, which are sometimes mistaken for pregnancy, the abdomen is protuberant and more or less tympanitic, owing to abnormal contraction of certain of the abdominal muscles and particularly of the diaphragm.

3. A peculiar thrill or sense of fluctuation on percussion. This is elicited by laying the left hand flat on the side of the abdomen and then tapping abruptly, but gently, on the other side with the fingers of the right hand. This thrill is always most decided when the quantity of liquid accumulated is great, and when the abdominal wall is thin and tense; but even a few

ounces may be detected by skilful hands. In this case, however, you must not expect to get the thrill propagated from one side of the abdomen to the other, but you must apply the fingers of the left hand over the upper margin of the part that is dull on percussion, and tap on the dull part a few inches below with the fingers of the right hand.

4. Pressure will sometimes give unmistakable evidence of fluid in the abdomen. If pressure be made with the tips of the fingers suddenly and perpendicularly to the surface, you will frequently experience a sensation of the displacement of liquid and of your fingers coming in contact with some solid body, such as an enlarged liver or spleen, or a tumour.

5. When the quantity of fluid is great, it will interfere with the proper action of the diaphragm and abdominal muscles, and cause more or less dyspnœa and thoracic breathing.

6. In cases also where there is a large accumulation of liquid the patient's head is often thrown back in standing or walking, to balance the body. The same gait is constantly observed in the advanced stage of pregnancy.

7. Mere accumulation of liquid in the abdomen, by compressing the renal and iliac veins, may give rise to albuminuria and anasarca of the legs, but these are important characters to which we shall return presently.

CONDITIONS SIMULATING FLUID IN THE PERITONEUM.

When the characters now enumerated are present, you may be perfectly sure that you have to deal with a collection of fluid in the abdomen, but they are not sufficient to enable you to say whether the fluid be in the peritoneal cavity or in a distinct cyst, and this is the next point which you must always proceed to determine. Fluid in the peritoneum is most readily simulated by: 1, an ovarian cyst; 2, a hydatid tumour; 3, a large cyst attached to the kidney; 4, a distended urinary bladder; or, 5, a pregnant uterus: and in practice it is very necessary that you should avoid confounding with it any of these conditions.

1. *An Ovarian Cyst.*

From its great frequency this is the condition most apt to be mistaken for ascites. As long as an ovarian tumour is small, its outline can be felt through the abdominal parietes and the diagnosis is easy. The difficulty arises when the cyst is very large and appears to fill the abdomen.[1] Even then, howeve is readily distinguished from ascites by the following charact most of which also serve to distinguish ascites from other well as ovarian, cysts in the abdomen containing fluid :

1. In ascites the fluid, being free to move about among bowels, always gravitates, and the intestines containing float on the surface, whatever be the position of the pati Consequently, when the patient lies on his back, there will dulness on percussion in the flanks and a clear tympan

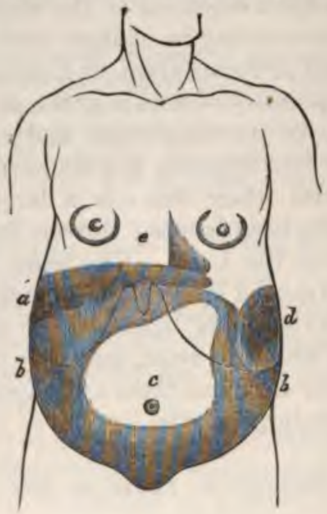

Fig. 30 illustrates the percussion-sounds over the abdomen in a case of ascites cirrhosis of the liver.

a. Dulness of contracted liver. *b*. Fluid in peritoneum causing bulging of the flanks. *c*. Tymp intestines. *d*. Enlarged spleen. *e*. Heart.

circular space of greater or less extent around the umbili generally more above the umbilicus than below it, inasmuch patients lie mostly with the shoulders more elevated than pelvis. When the patient lies on his right side, this cl space will shift to the left; and when he lies on the left side will shift to the right. Even small amounts of fluid may detected by making the patient rest upon his elbows and kn when the fluid will gravitate to the umbilicus. In any sit tion, the dulness on percussion will often disappear at its margin on deep pressure. But in ovarian dropsy the cyst ascends in front of the intestines, which are prevented from coming in front of it by the mesentery, and which are pressed back by the tumour against the spine. Accordingly, if there be any tympanitic resonance, it will be in one or both flanks, or in the epigastrium, and the umbilical region will be dull, while the relative position of the clear and dull spaces will not vary with the posture of the patient. These important differences between ascites and ovarian dropsy are illustrated in the annexed diagrams (figs. 30 and 31).

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[1] These remarks apply only to those cases where the tumour is entirely or m composed of one cyst. Multilocular ovarian cysts are readily distinguished ascites by their uneven surface, greater hardness and resistance on pressure, and b comparatively obscure fluctuation.

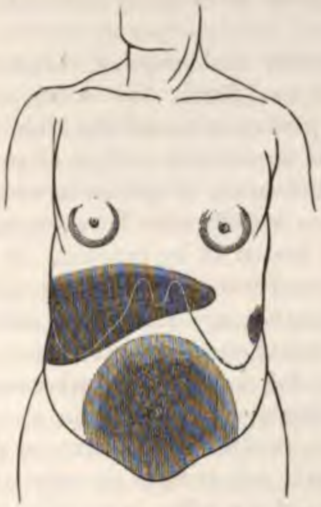

Fig. 31 illustrates the percussion-sounds over the abdomen in a case of tumour of the left ovary. The dulness of the tumour occupies the centre of the abdomen, which on either side is tympanitic.

2. In ascites the swelling of the abdomen is uniform and symmetrical from the first, and when the patient lies on his back, the weight of the fluid causes a bulging on either side and gives an appearance of increased breadth to the trunk. But in ovarian disease the swelling commences in one flank, and for a long time is more on one side of the abdomen than on the other; and at this stage also, when the hand is pressed back through the abdomen, so as to feel the lower part of the spine, the tumour may be felt to pass down into the pelvis. When large, although it may appear to fill one side of the abdomen as much as the other, it bulges forwards rather than laterally.

3. In ascites the distances between the umbilicus and the pubes and sternum maintain their normal ratio, the navel being about an inch nearer to the pubes than to the sternum; but in the case of ovarian tumour this ratio is often reversed. In the latter also, but never in ascites, the distance between the umbilicus and the crest of the ilium may differ on the two sides, being greater on the side from which the tumour has sprung. In ascites, when the patient is recumbent, the greatest girth of the abdomen is usually at the umbilicus or a little above this; but in ovarian tumour it is more commonly an inch or two below this.

4. In both ascites and ovarian tumour there is often obliteration of the umbilicus, but in simple ovarian tumour there is never any protuberance of the umbilicus, as is common in ascites. This is sometimes a sign of some importance as indicating the supervention of ascites on ovarian tumour.

5. There is often a difference between ascites and ovarian dropsy in the fluid drawn off by tapping. In ascites it is either a clear, straw-coloured water, having a specific gravity of about 1015, and containing a large quantity of albumen, or, if there has been any inflammation of the peritoneum, the fluid may be turbid and contain flakes of lymph; whereas the fluid from an ovarian cyst, although sometimes thin and almost colourless like that of ascites, is often of a glutinous consistence or of a brownish or chocolate colour from the admixture of blood.

By attention to these rules, you will seldom have much difficulty in distinguishing between ascites and ovarian dropsy. There are, however, certain sources of fallacy in the diagnosis which it is necessary to keep in view.

a. When the quantity of fluid in the peritoneum is very great, the mesentery may not be broad enough to allow the intestines to float to the surface, and consequently there may nowhere be any tympanitic sound elicited on percussion of the abdomen.

b. The intestines in ascites may be prevented from floating by old adhesions or by a diseased omentum binding them down to the spine. In cases, for instance, of sub-acute or chronic peritonitis, the intestines may be matted together and bound down to the back of the abdomen by old adhesions, while fluid collects between them and the abdominal wall in front (see Case CLII.).

c. In very rare cases an ovarian tumour has a coil of bowel stretched over it, which may yield a tympanitic percussion-sound.

d. An ovarian cyst may contain air as well as fluid, owing to decomposition of its contents after tapping, or to its having effected a communication with some portion of the bowel, as in Case CXLVIII. Under these circumstances you may have an ovarian cyst with tympanitic percussion-sound at the umbilicus.

e. Occasionally there is a concurrence in the same person of ascites and ovarian tumour. In this case, when the patient is supine, there is dulness on percussion at the umbilicus as well as in the flanks; but if he lie on the side from which the ovarian tumour springs, the percussion over the opposite side may be tympanitic. By pressing suddenly also with the points of the finger on the abdomen, in the manner already described, you may experience a sensation of displacing fluid, and at the same time of impinging upon an elastic tumour.

2. *Hydatid Tumour.*

It is not often that a hydatid tumour fills the abdomen to such an extent as to be readily mistaken for ascites, but occasionally a mistake of this sort is apt to arise. You will remember that in a former lecture (Case XXXVIII., p. 122) I related to you the case of a girl of 15, in whom a hydatid tumour springing from the liver caused such an enlargement of the abdomen as seriously to embarrass the function of respiration and threaten death from asphyxia—which was only temporarily averted by drawing off 248 oz. of fluid by paracentesis, and where the real nature of the case was only disclosed on post-mortem examination. Such a case, however, ought to be distinguished from ascites by the following characters:

1. By the swelling being in the first instance unsymmetrical, or confined to one portion of the abdomen, generally the hepatic region, before it becomes general. Most commonly a hydatid cyst commences in the hepatic region and grows downwards, but the possibility of its originating in the pelvis and growing upwards must be kept in view.[1]

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[1] See, for instance, a case recorded by Dr. Habershon in the Pathological Transactions (vol. xi. p. 155), where the tumour reached as high as the umbilicus and exactly resembled a distended urinary bladder; another reported by Mr. Bryant, where it reached as high as the scrobiculus cordis (Path. Trans. vol. xvii. p. 278); and a third reported in my paper on Hydatid Tumours in the Edin. Med. Journ. for

2. By the tympanitic portion of the abdomen not always being most elevated whatever posture the patient assumes. In the case already referred to there was tympanitic percussion in both flanks, while the anterior portion of the abdomen was dull and fluctuating.

3. By the fluid drawn off being clear and limpid, strongly impregnated with common salt, but devoid of albumen or urea (see p. 61).

In those rare cases already referred to (pp. 56, 69) where ascites coexists with hydatid tumour, the difficulty of diagnosis will be considerable.

3. *A Renal Cyst.*

A cyst attached to the kidney occasionally acquires such dimensions as to cause great enlargement of the abdomen. Not long ago you had an opportunity of seeing a case of this sort (see Case VIII. p. 27), where a cyst of the right kidney contained upwards of 200 oz. of fluid. However large such a cyst may be, it is always easily distinguished from ascites:

1. By the signs of fluid being limited to one side of the abdomen, and by the tympanitic intestines being pushed over to the other, in whatever posture the patient lies.

2. By there being often a history of some injury to the kidney years before.

3. By there being often a history of hæmaturia or albuminuria, or of some symptoms of urinary irritation. It is important, however, to remember that enormous renal cysts may exist independently of any urinary symptoms (see p. 27).

4. The fluid obtained by tapping may contain urea, and on adding nitric acid to the alcoholic extract crystals of nitrate of urea would be produced. Mr. Stanley has recorded two cases of this sort where the fluid of the cyst contained urea.[1] This character, however, although of value in pointing to the origin of a circumscribed cyst in the abdomen, would not distinguish the case from ascites, for in dropsy from diseased kidneys the fluid that collects in the peritoneum and elsewhere has been

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Dec. 1865, Case X. In most of these cases the tumour interferes sooner or later with micturition.

[1] An Account of two Cases of Rupture of the Ureter or Pelvis of the Kidney. Medico-Chirurgical Transactions, 1844, vol. xxvii. p. 1.

often shown to contain urea in large quantity. Moreover, we have already found that urea is not always present in the fluid of renal cysts (see p. 27).

4. *A distended Urinary Bladder.*

You may think it very improbable that a distended urinary bladder could ever be taken for ascites, or indeed for anything else than a distended bladder, but such a mistake has more than once been committed, and there can be no doubt that the bladder in rare cases becomes so enormously distended as to fill a great part of the abdomen, and simulate ascites or cystic tumours. There is the authority of Sir Everard Home for the fact that John Hunter once actually tapped a distended bladder in the belief that the disorder was ascites; and from my own experience I am inclined to agree with Sir Thomas Watson in thinking that mistakes of this sort are not uncommon in private practice.[1] They are all the more likely to occur from the fact that the patient often passes a fair amount of normal urine. I show you here a bottle of urine obtained by paracentesis from a patient, who was believed by one of the most experienced surgeons of the present day to be suffering from a large hydatid tumour of the abdomen. You may judge of the size of the bladder in this case, when I tell you that 480 oz. of fluid were drawn off by a small trocar introduced midway between the umbilicus and the sternum. The case is one of such importance in diagnosis that I shall not be wasting your time in relating it to you in detail (see Case CXLIX.). Case CL. is another remarkable illustration of the same mistake. I may refer also to a case which occurred in my practice a few years ago, where a sacculus of the bladder in a man aged 68, became so enormously dilated as to form a large tumour in the right iliac region, which compressed the femoral vein and caused thrombosis of this vessel with painful swelling of the leg.[2] Cases of distended bladder will be distinguished from ascites:

1. By there being dulness at the umbilicus and displacement of the bowels upwards and laterally.
2. By there being in the earlier stages a central circum-

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[1] Lect. on Practice of Physic, 5th edit. vol. ii. p. 445.

[2] In this case also the patient passed normal urine freely, and seemed to have no urinary symptoms. The cyst was tapped midway between the pubes and the spine of the ilium and 12 oz. of urine drawn off; the operation was followed by no bad consequence. The case is fully reported in the Pathological Transactions (vol. xiv. p. 133).

scribed tumour containing fluid, and growing upwards from the pubes.

3. By there having been in most cases at some former period symptoms of retention or of some urinary disturbance, even though such symptoms may be absent subsequently when the patient first comes under notice.

4. Cases of this sort are most common in old men with large prostates, where an ovarian cyst, which most closely simulates the characters of a distended bladder, may be excluded from the diagnosis, and whenever from the circumstances any doubts exist as to the enlargement being due to the bladder, they may be solved by introducing a long prostatic catheter and pushing it well up. With an ordinary catheter only a few ounces of urine may be obtained, and no impression may be made on the tumour, for, as was long ago shown by Deschamps,[1] the bladder in these cases bends over upon itself so that the greater part of the viscus is shut off from the neck, which is also distended so as to hold a few ounces of the urine.

5. *A Pregnant Uterus.*

Although from the time of Queen Mary there have been many celebrated instances of abdominal dropsy being mistaken for pregnancy, and *vice versâ*, it would be an unjustifiable error for a medical man at the present day to mistake a solid enlargement of the uterus, accompanied by all the constitutional symptoms and local signs of the pregnant state, for dropsy of the peritoneum.

CAUSES OF FLUID IN THE PERITONEUM.

But supposing you have decided that there is really fluid in the peritoneal cavity, the next point to determine is its source or cause. It may be due to: I. Acute Peritonitis; II. Tubercular Peritonitis; III. Chronic Peritonitis; IV. Cancer of the Peritoneum; V. Colloid disease of the Peritoneum; or, VI. Simple Dropsy.

I. *Acute peritonitis* is distinguished by:

1. Its rapid course.

2. The symptoms of abdominal inflammation—pyrexia,

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[1] Traité de l'Opération de la Taille, tom. i. p. 224. This is a source of fallacy in diagnosis on which, thirty years ago, Mr. Syme was in the habit of strongly insisting in his clinical lectures.

with small, rapid pulse, shrunken features, clammy sweats, and great tendency to collapse; vomiting; acute pain and tenderness of the abdomen; thoracic breathing; and legs drawn up.

3. The quantity of fluid thrown out into the peritoneum is usually small, and is often insufficient to produce a distinct sense of thrill or fluctuation.

II. *In Tubercular peritonitis* the tendency is to the formation of firm adhesions of the abdominal viscera to one another and between them and the abdominal wall, without any accumulation of fluid. The abdomen in these cases is usually retracted. Occasionally circumscribed collections of fluid form between adjacent coils of bowel (from tubercular ulcers of the mucous coat ending in perforation, or from mere softening of tubercular matter), and acquire such a size as to cause a bulging of the abdominal wall. In other cases fluid in considerable quantity collects in the peritoneal cavity as the result of slight tubercular peritonitis, probably associated with deposit of tubercle in the portal lymphatics which press upon the portal vein (see Case CLVI.). I have met with at least two cases of this sort in which there has been a simultaneous collection of fluid in one of the pleural cavities, and similar cases have been observed by Leudet.[1] What is noticeable also about these cases is that the fluid is often absorbed, especially in the case of children, and the patient recovers. Not unfrequently in tubercular peritonitis, the fluid is purulent, and sometimes it points at the umbilicus, and more than once I have known the patient recover after its evacuation. One remarkable instance is on record in which tubercular peritonitis was mistaken for ovarian disease; after 18 pints of fluid had been removed by tapping, Mr. Spencer Wells laid open the peritoneum, which was found to be studded with myriads of tubercles, while the intestines were matted together and bound down towards the back and upper part of the abdomen. The cavity was emptied and the wound closed. After a sharp attack of peritonitis the patient recovered; at the end of four years she married, and six years later she was stout and well.[2]

The symptoms of tubercular peritonitis are often obscure, but it may generally be recognised by the following characters:

1. The presence of hectic fever with emaciation and night-sweats.

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[1] Clin. Méd., Paris, 1874, p. 506.

[2] Dr. Hilton Fagge. Guy's Hosp. Rep. 1875, vol. xx.

2. The concurrence of signs of tubercle in other parts of the body.

3. Diarrhœa is not uncommon.

4. In some cases there are pain and tenderness, but not acute, in the abdomen.

5. Occasionally hard masses of indurated omentum can be felt.

III. *Chronic Peritonitis,* independent of either tubercle or cancer, is not very uncommon. Its pathology has not yet been satisfactorily worked out. In some cases the inflammation seems to start from one of the subjacent viscera, and in others the patient is of broken-down constitution—very often the subject of Bright's disease. The intestines in these cases become matted together and tied down to the spine, and fluid accumulates in considerable quantity between them and the abdominal wall in front (see Case CLII.). These cases are distinguished as follows:

1. There is symmetrical and rarely very great enlargement of the abdomen.

2. There is distinct fluctuation, but nowhere tympanitic percussion-sound, except perhaps at the epigastrium.

3. There is in most cases slight fever with pain and tenderness of abdomen and thoracic breathing—the symptoms in fact of acute peritonitis in a very modified form. Occasionally the symptoms at the commencement are those of acute peritonitis.

4. The diagnosis may be assisted by there being an absence of other causes of fluid in the peritoneum, such as disease of the heart, liver, or kidneys.

There are certain cases which seem to constitute a connecting link between inflammation and dropsy of the peritoneum, where the attack commences with symptoms of subacute inflammation, but subsequently takes on more the characters of dropsy. These attacks are most common in children and in women, and in the latter are often associated with some inflammatory condition of the pelvic organs, or the leakage of an ovarian cyst. The dropsical effusion in these cases often rapidly disappears under treatment. I have met with persons in advanced life who have had attacks of this sort when children, for which a large quantity of fluid has been drawn off by paracentesis, and who have made a complete and permanent recovery. It is possible that the inflammatory process in the first instance causes obstruction of certain of the mesenteric veins, as the result of which dropsical effusion takes place, which disappears on the removal of the obstruction, or on the enlargement of collateral veins. To this class probably belong the cases referred to by Sir Thomas Watson under the designation of *active ascites*, where fluid is rapidly thrown out into the peritoneum after exposure to cold and wet, without fever or any sign of inflammation, and independently of any disease of the liver, heart, or kidneys, and where after a short time it is reabsorbed.[1] Professor Leudet of Rouen also has within the last few years published a number of interesting observations, illustrating the curability of ascites resulting from subacute inflammation of the peritoneum.[2] Case CLVIII. appears to be an example of this form of dropsy.

IV. *Cancer of the Peritoneum.*—It is not often that the peritoneum is the seat of cancer, but cases are every now and then met with, where cancer commences in, and at death is still limited to, the peritoneum. The peculiarity of these cases is that there always is more or less pain, with the effusion usually of a large quantity of fluid into the peritoneal cavity, often necessitating paracentesis to avert asphyxia, and in this respect presenting a striking contrast to tubercular peritonitis. You ought always to suspect cancer of the peritoneum when you meet with pain and effusion in the abdomen in aged persons. Six cases answering to this description have come under my notice, and in two you will observe that the patients were under forty years of age. Three of the cases, Jane O., aged 51, Catherine H., aged 38, and Mary Anne P., aged 48, I shall presently describe to you in detail (Cases CLIII. to CLV.). The fourth was the case of a rickety female, aged 78, who died in Middlesex Hospital on Oct. 10, 1860, whose abdomen was enormously distended with many quarts of flaky fluid, and the intestines studded on their peritoneal surface with numerous small nodules of cancer of about the size of split-peas. Three months before death she had been attacked with vomiting and abdominal pain, accompanied by emaciation, and followed after six weeks by ascites, rapidly increasing till death. The fifth was the case of a man aged 39, who died in Middlesex Hospital on Feb. 19, 1861, and in whose body I found appearances precisely similar to those in the fifth case. His illness had commenced 2½ months prior to death with pain and tenderness of the abdomen, followed by rapidly increasing ascites. The sixth was that of a bargeman, aged 54, who was under my care in Middlesex Hospital for several weeks in the autumn of 1863, whose illness commenced three months prior to death with an injury to the abdomen, and in whose peritoneum there was also a large accumulation of slightly turbid and flaky fluid, with nodules of medullary cancer scattered over the peritoneal surface of the different viscera.

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[1] Lect. on Med. 5th ed. ii. 439.

[2] Clin. Méd. Paris, 1874, p. 516.

The characters by which you will recognise these cases are the following:

1. There are the phenomena of the cancerous cachexia, and perhaps a family history of cancer.

2. There is an accumulation of a large quantity of fluid in the peritoneum, independently of disease of the liver, heart, or kidneys.

3. There is more or less fever, with great emaciation, vomiting, constipation, and severe pain and tenderness of the abdomen, approaching those of acute peritonitis. From acute peritonitis, however, the affection differs in the extensive effusion of fluid: in ordinary acute peritonitis also the tendency to collapse is greater and the course more rapid.

4. It is not often that the nodules of cancer are large enough to be felt through the abdominal parietes, but the diagnosis will sometimes be assisted by discovering a mass of thickened omentum in the umbilical region.

V. *Colloid disease* occasionally causes great enlargement of the abdomen without any fluid in the peritoneum. You will find two cases of this sort recorded in the Pathological Transactions by Dr. Vanderbyl and Dr. O'Connor.[1] I show you here specimens of the colloid disease from the former case, which proved fatal in this (Middlesex) hospital. Quite recently, you have had a similar case under your own observation.[2] These cases may often be recognised by the abdomen becoming distended by a firm nodulated mass, glueing all the organs together, dull on percussion everywhere in front, but leaving a tympanitic space in either flank. But in some cases colloid disease, like ordinary cancer, leads to a large accumulation of fluid in the peritoneum either from exciting peritonitis or from the rupture of some of the colloid cysts and discharge of their contents, and paracentesis has been necessary to relieve the breathing. (Case CLXIV). The symptoms in these cases are very similar to those of primary cancer of the peritoneum (Case CLXIII.). There is pain with tenderness in the abdomen, often preceding the signs of fluid for several weeks and always accompanying them; in most cases there is a certain degree of pyrexia and vomiting; but the swelling is much greater than in ordinary acute peritonitis. From the first there is rapid emaciation, and the case usually terminates fatally within six months of the commencement of the symptoms. As in cancer also, the disease is chiefly met with in middle and advanced life; of nine cases reported in the Pathological Transactions, the ages varied from 37 to 75. But from cancer of the peritoneum colloid disease differs in these respects:

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[1] Vol. ix. p. 207, and vol. xiii. p. 90.

[2] Emma Barrett, aged 32, adm. into St. Thomas's Hosp. July 10, and died Sept. 5, 1875.

1. Even when the abdomen is very large, fluctuation is often very indistinct.

2. The fluid obtained by tapping is gelatinous, and sometimes so viscid that it will not flow through the cannula.[1] At other times it is thin, but turbid, and contains blood and cells of the colloid matter.

3. Hard irregular masses or distinct tumours can sometimes be felt through the abdominal wall.

4. Occasionally the patient voids per anum a gelatinous or slimy fluid, like white of eggs.[2]

VI. *Simple Dropsy of the Peritoneum.*—The causes of fluid in the peritoneal cavity already referred to being excluded, its presence must be due to simple dropsical effusion. This has the following characters:

1. There are the signs of fluid in the peritoneal cavity already enumerated (see p. 434).

2. There is an absence of persistent pain and tenderness of the abdomen, and usually of pyrexia.

Simple dropsy of the peritoneum may have a threefold origin. It may be due to:

1. Diseases of the kidneys.
2. Intra-thoracic diseases.
3. Diseases of the liver or portal vein.

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[1] This happened in a case reported by Dr. Dickinson in the Pathological Transactions, vol. xii. p. 92.

[2] Two cases are recorded in the Pathological Transactions where this occurred—one by Dr. Quain (vol. iii. p. 319), and the other by Dr. O'Connor (vol. xiii. p. 90).

Some writers have described a form of ascites which they have designated *idiopathic*, to imply that it is independent of any organic lesion either in the abdomen or elsewhere. Inasmuch as these cases usually recover, it may be difficult to determine their real pathology, but most of them are probably either tubercular (see p. 443) or inflammatory (see p. 444) in their origin.

I. DROPSY OF THE PERITONEUM FROM DISEASES OF THE KIDNEYS.

The diseases of the kidneys which are most likely to induce dropsy are: *a*, acute nephritis, occurring as a sequel of scarlatina or from exposure to cold and wet, when the kidneys are hypertrophied and hyperæmic; *b*, the so-called large white kidney, when the cortex is pale and hypertrophied and the uriniferous tubes crammed with granular epithelium, and which is often merely an advanced stage of acute nephritis, but may also be developed independently as the result of a chronic inflammatory process; *c*, the fatty kidney, where the organ is also large and pale, but the secreting cells are loaded with oil. Both clinical experience and post-mortem examinations make it probable that the fatty kidney is usually preceded by the large white form of the disease. Either of these three diseases of the kidneys may give rise to ascites, which has the following distinguishing characters:

1. There is anasarca of the subcutaneous areolar tissue, which is *general* from the first, and is often noticed first in the face.

2. There is almost always evidence of fluid in the other serous cavities—the pleuræ and pericardium, as well as in the peritoneum.

3. The urine is scanty, turbid, or smoky, and contains a large quantity of albumen and possibly blood. Under the microscope you will usually find renal epithelium and casts of the uriniferous tubes varying in their character according to the particular disease in the kidney—epithelial, blood, and hyaline casts in acute nephritis; granular epithelial casts in the large white kidney; and oil-casts in the fatty kidney.

4. There is a pallor and pastiness of the countenance which is almost pathognomonic.

5. There is a tendency to uræmia and the typhoid state,

indicated by a dry brown tongue, fetid breath, loss of memory and restlessness, delirium, coma and convulsions.

All of these clinical characters you had an opportunity of studying in the case of James S——, who was lately under my care in Middlesex Hospital (Case CLX.).

In forming a diagnosis it is necessary to remember that most renal diseases predispose to inflammation of the serous membranes and that thus the symptoms of peritonitis may concur with those of renal dropsy, and also that the urine may contain albumen although the ascites is independent of disease of the kidneys (see pp. 282, 288).

Two common forms of chronic kidney-disease are rarely attended by dropsy and still more rarely by ascites. In the contracted, granular, or gouty kidney there is usually little or no dropsy at any stage of the malady; while in the waxy, or amyloid kidney anasarca rarely shows itself until shortly before the fatal termination, and even then is seldom excessive (see p. 33). Both of these diseases of the kidneys are marked by the secretion of an increased amount of urine and by a comparative absence from it of uriniferous casts; but in the contracted kidney the specific gravity is low out of all proportion to the extent of dilution, the albumen is scanty or even altogether absent, there is often a history of gout, and there is a marked tendency to uræmia; while in the case of the waxy kidney the lowness of the specific gravity of the urine is not more than may be accounted for by its dilution, the amount of albumen in it is large, and there is little tendency to uræmic symptoms, but in most cases there are the signs of waxy enlargement of the liver and spleen, exhausting diarrhœa, and a history of protracted purulent discharge (see p. 34).

II. DROPSY OF THE PERITONEUM FROM DISEASES IN THE CHEST.

Ascites is often a consequence of obstruction to the general circulation from various diseases of the chest, and especially from morbid conditions of the valves of the heart. Diseases of the mitral valve and tricuspid incompetence, whether secondary to mitral disease or to chronic bronchitis and emphysema, are more likely to cause dropsy and ascites than lesions of the aortic valves. A tumour compressing the inferior vena cava above its junction with the hepatic vein may lead to a like result.

Fluid in the peritoneum due to diseases obstructing the circulation of blood through the heart or lungs has the following characters:

1. Before there is any ascites there is anasarca, commencing in the feet and gradually proceeding upwards; and even when the belly becomes swollen, the swelling of the legs is large out of all proportion to the ascites.

2. There is a history of dyspnœa before any swelling shows itself in the abdomen. In extensive ascites, whatever be its cause, there is usually more or less dyspnœa from the pressure of the fluid interfering with the action of the diaphragm and abdominal muscles. The peculiarity of the dropsy from intra-thoracic disease is that the dyspnœa precedes the ascites and is distressing out of all proportion to the extent of the dropsy.

3. Intra-thoracic diseases which cause dropsy give rise at the same time to more or less lividity of the lips, face, and extremities.

4. There are the physical signs and symptoms of valvular disease of the heart or of old disease of the lungs. Even when the primary seat of obstruction is in the heart, there will be evidence of congestion or œdema of the lungs, or of bronchitis, pulmonary apoplexy, etc.

5. In the case of a tumour compressing the inferior vena cava there will be: *a*, the physical signs of the tumour; *b*, indications of its pressure upon other parts, such as the œsophagus or lung; *c*, a more rapid development and increase of anasarca than in ordinary cases of cardiac obstruction; and *d*, great enlargement and tortuosity of the superficial veins of the chest and abdomen.

III. DROPSY OF THE PERITONEUM FROM DISEASES OF THE LIVER OR OF THE PORTAL VEIN.

The ascites which results from obstruction to the circulation through the trunk of the portal vein, or through its ramifications in the interior of the liver, is that with which at present we are more immediately concerned, and we shall therefore consider not merely the characters of the ascites from portal obstruction in general, but also those which pertain to the several causes of this obstruction. And in the first place, with regard to the distinguishing characters of the ascites from any cause of portal obstruction. They are as follows:

1. The dropsy in uncomplicated portal obstruction com-

mences in the abdomen. The legs are only affected secondarily and in consequence of the pressure of the ascitic fluid on the inferior vena cava. There are, however, exceptions to this rule. Patients will tell you occasionally that they have noticed slight swelling in the legs as soon as the abdomen began to enlarge; but in these cases there is this to be noted, that the swelling of the legs is slight out of all proportion to that of the abdomen, and that when the patient takes to bed it diminishes or entirely disappears, while the ascites remains stationary or increases. Outgrowths from the liver, however, such as occur in cancer and cirrhosis, which compress both the portal vein and the inferior vena cava in the notch at the posterior part of the liver, may cause permanent dropsy both of the legs and abdomen. And lastly, there are those cases of chronic induration of the liver with portal obstruction which are secondary to diseases in the chest obstructing the general circulation, to which I have formerly adverted (p. 275); in such cases the ascites will be preceded by dropsy of the legs, and the addition of portal to general obstruction is not likely to be suspected unless there be a great preponderance of the dropsy in the peritoneum.

2. There is no sign of dropsy in the face, arms, or upper part of the trunk.

3. Dyspnœa often accompanies the swelling when this is great, but does not precede it, except in the one condition already referred to (p. 283).

4. Albuminuria is absent if there be no concurrent kidney-disease. In reference, however, to this point, there is a source of fallacy. The ascites itself, when extensive, may in consequence of the pressure of the fluid on the renal veins lead to the appearance of albumen in the urine, the albuminuria ceasing on the withdrawal of the pressure by the operation of paracentesis. At the same time you must remember that albuminuria will not admit of this explanation, unless the ascites be so great as to cause considerable tension of the abdominal parietes. The concurrence of puffiness of the face, or of pitting of the arms or upper part of the trunk, or the presence of granular or oil-casts in the urine would also leave little doubt as to the existence of independent renal disease.

5. In hepatic dropsy the urine is usually scanty, high-coloured, and loaded with pigmentary matter and lithates. The absence of these signs would be a strong argument against either cancer or cirrhosis of the liver.

6. The ascites is accompanied by other indications of portal obstruction, such as enlargement of the spleen, enlargement and tortuosity of the superficial veins of the abdomen, hæmorrhoids, gastro-enteritis and hæmorrhages from the stomach and bowels, which I have already described to you in a former lecture (see p. 279). The enlargement of the superficial veins of the abdomen is due to the collateral circulation established through the hæmorrhoidal plexus between the branches of the portal vein and those of the vena cava inferior, but is far from being a certain indication of the existence of portal obstruction. The same appearance is constantly observed in the ascites from disease of the heart, and sometimes as the result of pressure of a tumour or even of a large quantity of ascitic fluid on the inferior vena cava; but in these cases there will be usually also a varicose state of the veins of the legs, although in the case of compression of the inferior cava this may be in great measure prevented by great enlargement of the vena azygos. It must not be forgotten also that the epigastric veins, without being at all enlarged, may be rendered more visible in great ascites owing to the stretching of the abdominal walls and the absorption of the subcutaneous fat.

7. There is little or no pyrexia and no tenderness of the abdomen, except over the liver.

8. Other indications of the existence of hepatic disease, such as enlargement, contraction, nodulation, or tenderness of the liver, jaundice, flatulence, &c., will often assist the diagnosis, but you must remember that all these signs may be absent or indefinite.

9. The fluid obtained by paracentesis is a clear, straw-coloured serum, having a specific gravity of from 1012 to 1016, and containing a large quantity of albumen, but no urea, blood, nor inflammatory products.

In the next place we have to inquire what are the diseases of the liver which are most likely to occasion ascites. And first it must be remarked that there are some morbid conditions of the liver which very seldom give rise to it. The fatty liver (see p. 48) and simple hypertrophy of the liver (p. 53), when uncomplicated, never cause it, and it rarely results from mere congestion (p. 133), unless this depend on mechanical obstruction of the circulation in the chest. Abscess (pp. 165, 184) and hydatid tumour (pp. 56, 102) of the liver do not lead to the presence of fluid in the peritoneum, except in rare cases, from the direct pressure of the tumour on the trunk of the portal vein, from the bursting of the cyst, or through the intervention of peritonitis.

Fluid in the peritoneum is a more common accompaniment of the waxy or amyloid liver (p. 33) than of any of the maladies just named, but even here it is rare. Frerichs noted it in only 8 out of 23 cases, and in 4 of the 8 cases it was due to the supervention of acute peritonitis.[1] When its origin is not inflammatory, it is probably due to enlarged waxy lymphatic glands in the fissure of the liver pressing upon the trunk of the portal vein, or to the concurrence of peritonitis or cirrhosis (pp. 33, 47). The freedom from ascites in waxy liver is accounted for by the fact that it is the branches of the hepatic artery, and not of the portal vein, which are implicated in the disease. Fluid in the peritoneum from waxy liver will be recognised by the following characters (see also p. 31) :

1. Uniform (but in very rare instances nodulated, pp. 32, 47), solid, painless enlargement of the liver.
2. Enlargement of the spleen.
3. A large quantity of albumen in the urine, with little or no anasarca, and those other characters of the urine which I described to you in my lecture on waxy enlargement of the liver (p. 34). Albuminuria, however, is not necessarily present in cases of waxy disease. Since my lecture on the subject was delivered, you have seen two cases in the wards, where there was considerable waxy enlargement of both liver and spleen, but where the kidneys were so slightly implicated that the urine contained no albumen.
4. Great anæmia.
5. A history of disease of the bones, or joints, suppurating sores, phthisis, or constitutional syphilis.

The diseases of the liver which most commonly give rise to portal obstruction with ascites are :

1. Cirrhosis and the other forms of interstitial hepatitis.
2. Cancer of the liver.
3. Peri-hepatitis.
4. Thrombosis or obstruction of the trunk of the portal vein.

Practically, it is chiefly with the two first that you will have to deal, as the others are of much rarer occurrence.

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[1] Diseases of the Liver. Syd. Soc. Ed. vol. ii. p. 179.

compressing force be great, the vessel may be flattened and its walls brought into apposition; more commonly the compression acts by retarding the circulation and favouring coagulation.

d. Lastly, there are some cases of thrombosis of the portal vein which seem to result from mere weakness of the circulation, or from an unusual disposition of the blood to coagulate.

The symptoms of thrombosis of the trunk of the portal vein are those already described to you (p. 279) as resulting from obstruction or obliteration of the ramifications of the vessel in chronic atrophy, but in an exaggerated form. They are:

1. The rapid development of extreme ascites, often necessitating paracentesis to avert asphyxia, and returning immediately after the operation.
2. Rapid enlargement of the veins of the abdominal wall, which resemble large cords.
3. Urgent vomiting and diarrhœa.
4. Copious hæmorrhage in many cases from the stomach and bowels. Occasionally this is the first and chief symptom, and the patient dies from syncope before there is time for ascites &c.
5. Great enlargement of the spleen.
6. Obstruction of the trunk of the portal vein in most cases is speedily fatal, but if it be not, it leads to atrophy of the liver.[1] A contracted liver may thus be either the cause or the consequence of portal obstruction.

I have thus endeavoured to lay before you succinctly, and perhaps somewhat dogmatically, the distinguishing characters of fluid in the peritoneum according to its several causes. In practice, however, you must remember that there may be a concurrence of different causes in the same case. For instance, I have more than once pointed out to you that portal obstruction may be secondary to obstruction of the circulation in the heart or lungs, and I have also shown you that ascites may result from either of these causes independently of the other. Secondly, although, as we have seen, albuminuria may be induced by the ascites itself (p. 282), it is quite possible, and indeed not uncommon, to have disease of the kidneys concurrent with portal obstruction. Spirit-drinking, which is so fruitful a source of cirrhosis of the liver, contributes also to the development in many cases of nephritis and of the fatty kidney. Lastly, thrombosis of the trunk of the portal vein is often a result of obstruction of the ramifications of the vessel in the interior of the liver, and when this occurs, the symptoms of slight portal obstruction may be suddenly succeeded by those of complete obstruction.

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[1] See for instance a case reported by Dr. Dickinson in the Pathological Transactions, vol. xiv. p. 63.

The Treatment of Fluid in the Peritoneum.

The proper treatment for any case where there is fluid in the peritoneum must depend entirely on the cause of this condition, and you will therefore perceive the necessity which exists of being able to recognise the cause, or the predominant cause, in each particular case. It would be out of place on the present occasion, when I wish to direct your attention mainly to the diseases of the liver, to enter into a consideration of the proper treatment for the various maladies that may give rise to fluid in the peritoneum; and I have already described to you in some detail the appropriate treatment for the ascites and other symptoms resulting from portal obstruction. It is unnecessary to recapitulate the remarks on this matter, made in my lecture on Chronic Atrophy of the Liver (p. 285).

It may, however, serve to impress more durably on your minds the remarks which have now been made on the distinguishing characters of fluid in the peritoneum according to its several causes, and also the rules laid down in my systematic lectures on Medicine for the treatment of the various maladies from which ascites may arise, if I bring under your notice the particulars of a few cases which, with three exceptions, you have had an opportunity of watching in the wards.

Case CXLVIII. illustrated at first the points of distinction between fluid in an ovarian cyst and ascites or fluid in the peritoneum; but subsequently the physical signs were remarkably modified by the entrance of air into the ovarian cyst. A mistake in diagnosis was all the more likely to have been committed by a careless observer from the concurrence of albuminuria and general dropsy. The case is remarkable from its rare mode of termination, the ovarian cyst opening into the rectum. No similar case is reported in the entire series of the Pathological Transactions: in the fourteenth volume (p. 201) Dr. Bristowe has recorded a case where there was a communication between the rectum and an ovarian cyst, but in that case there was extensive tubercular ulceration of the bowel, and the perforation advanced from the bowel to the ovary. In the case of Elizabeth C—— you will remember that we were enabled to diagnose not merely the existence of the ovarian tumour, but also the fact of the cyst having burst into some portion of the bowel. After removal from the body, the walls of the cyst collapsed and shrivelled, so that no adequate idea of its size prior to bursting can be gathered from the preparation which I now show you.

CASE CXLVIII.—*Cystic Tumour of Ovary opening into Rectum—Entrance of Air into Ovarian Cyst—Atrophy of Right Lobe of Liver and complementary Hypertrophy of Left Lobe.*

Elizabeth C——, aged 37, adm. into Middlesex Hosp. Aug. 23, 1866. Had been married twice, but had only one child, still-born (1852), and never any miscarriages. Catamenia regular, last period having ceased day before admission. At age of 16 had been laid up for six weeks with scarlet fever, but did not know if she had dropsy. Ever since had suffered from pain in back, and for last eight years had been liable to general dropsy and attacks of erysipelas of face. Eighteen months before admission had first noticed a swelling in lower part of abdomen, which had been slowly increasing.

On admission abdomen considerably distended by a tumour rising above pubes and reaching to above umbilicus. This appeared to occupy a middle position in abdomen, but could be traced more readily into left side of pelvis than into right. Tumour was dull on percussion and distinctly fluctuating; behind it, in either flank, percussion yielded a tympanitic sound. Both lower extremities much swollen, œdematous, and tender, and face slightly puffy. Cardiac and respiratory signs normal, but areæ of hepatic and splenic dulness increased. Urine contained a considerable amount of albumen, and deposited epithelial and oily casts; sp. gr. 1016. Pulse 96 and feeble; occasional vomiting. On Aug. 25 patient began to have diarrhœa; motions contained blood, and there was considerable tenderness of abdominal tumour.

On Aug. 31 diarrhœa continued; no pain in defæcation; tongue clean and too red; breath very offensive.

On Sept. 10 no abatement of diarrhœa, notwithstanding free exhibition of astringents. Tongue dry and brown and breath extremely offensive. Patient was extremely prostrate, drowsy (from opium?), and occasionally delirious. No rigors nor sweating, and no diminution in size of tumour, girth of abdomen being same as at time of admission, (36 in.).

On Sept. 11 motions contained a quantity of pus, which continued to be passed for three days, and on Sept. 17 all signs of tumour had disappeared, percussion sound above pubes being equally tympanitic as in flanks.

After this little diarrhœa, but patient continued to sink, and died on Sept. 19.

At *autopsy* a thick layer of fat beneath skin. Heart and lungs healthy. Liver weighed 53 oz.; right lobe much atrophied and deeply lobulated, but left lobe enormously increased, being nearly three times size of right; structure appeared healthy. Spleen weighed 20 oz.; very firm, and presented typical characters and reaction of waxy degeneration. The two kidneys weighed together 18½ oz.; both smooth, pale-yellow and opaque; cortex greatly hypertrophied and secreting cells loaded with oil. On first opening abdomen, no tumour visible; intestines came down to pubes; but on raising a few coils, a collapsed cyst, about size of a cocoa-nut, was seen in situation of uterus. On further examination, this was ascertained to be a cyst of left ovary, which had emptied itself by an opening, size of a fourpenny-piece, into rectum 4 in. above anus; its walls were fibrous and about half an inch thick, and it contained a little dirty, very fetid pus. Sigmoid flexure of colon took a turn transversely to right side across upper part of tumour, to which it was firmly adherent; free end of appendix vermiformis also adhered to it. No ulceration of mucous membrane of rectum round opening into ovarian cyst.

The following very remarkable case illustrates the difficulty in diagnosis which may arise from great enlargement of the urinary bladder. In a female the physical signs would have at once suggested an ovarian tumour, but in a male they seemed only explicable on the supposition of a hydatid of the liver, and it was with this view that the surgeon under whose care the patient was had recourse to paracentesis. The great size of the swelling, the patient's statement that it commenced above the umbilicus, the fact of the greatest girth of the abdomen being 3 in. above the umbilicus, and the complete freedom from urinary symptoms, suppressed all suspicion of the bladder being primarily at fault. Such a case is not likely ever to occur in a female, and the lesson which it teaches is that whenever you find the physical signs which were present in this case, and especially when they occur in an aged male, the first thing to be done is to introduce a large prostatic catheter into the bladder and to push it well up. It is worth observing, however, that in this case the puncture was not followed by any extravasation of urine or sign of peritonitis, and that the immediate cause of death appeared to be passive hæmorrhage from the mucous membrane of the bladder, in consequence of the withdrawal of the urine.

Case CXLIX.—*Enlargement of Abdomen from a distended Urinary Bladder mistaken for a Hydatid Tumour of Liver—480 oz. of Urine drawn off by Paracentesis Abdominis.*

Mr. F——, a feeble-looking elderly gentleman, of small frame and spare habit, consulted an eminent surgeon, to whom I am indebted for these particulars, at beginning of June 1866. He stated that until three years before he had always enjoyed perfect health and been engaged in active pursuits. He then first noticed a swelling *above the navel*, which continued to increase, but caused him little inconvenience until beginning of previous March, when his breath became short and he began to lose flesh and strength and to get nervous. Early in May his left thigh and leg became swollen and pitted on pressure, but after two weeks swelling disappeared. For several weeks he had been troubled with frequent vomiting after food. He had nevertheless been able to ride for several hours a day up to time of his presenting himself for advice. His condition was then noted to be as follows:—'He presents appearance characteristic of abdominal disease. Face thin and cadaverous, but not jaundiced. Abdomen enormously enlarged, being occupied by a tumour of uniform surface and oval form, of which narrow end is uppermost. Ensiform cartilage and lower ribs on both sides elevated, and tumour appears to spring from beneath them and to fill up entire anterior portion of abdomen. Fluctuation is distinct throughout every part of it, but is particularly marked above umbilicus. Tumour is everywhere dull on percussion, but in both flanks, in whatever posture patient assumes, there is tympanitic percussion-sound. Abdomen is largest 3 in. *above* umbilicus, where its girth is 43 in.; distance from pubes to ensiform cartilage measures 21 in. A few blue veins are seen coursing over abdominal wall. Heart-sounds weak, but in other respects normal. Urine abundant; sp. gr. 1010; faintly acid, and with a slight trace of albumen. He has passed water three or four times a day, and once during night, always in a full stream and with perfect relief.' Patient always insisted that he had never suffered the slightest urinary symptoms nor trouble in micturating, but subsequently, after bladder had been tapped, he admitted that for some years he had made a practice of sitting down when he made water, because he had found that the urine 'came better' when he did so.

On June 5 abdomen was tapped with a fine trocar midway between umbilicus and ensiform cartilage. Twelve quarts, or 480 oz., of fluid drained away, the time occupied being two hours, and tumour slowly disappearing. The fluid was of a pale straw colour; sp. gr. 1010, feebly acid, slightly albuminous, and had a urinous odour. A portion sent to me for examination contained urea and a few blood-corpuscles.

June 6.—Patient has had a comfortable night, but has passed no urine since operation. Half a pint of *dark* urine drawn off by catheter in morning, and 1½ pint in evening. Abdomen is again large.

June 7.—Has had another good night and has no symptom of constitutional disturbance. A pint and a half of dark urine was drawn off in morning, and at 3 P.M. *six pints* more, and as this flowed away swelling again disappeared, and patient's belly became quite flat. Catheter was tied in but soon slipped out. During the hour occupied in emptying bladder patient felt very faint, and soon afterwards he had a rigor and fainted on getting up to try to micturate. Stimulants were given freely and fainting did not return, but he had a restless night and vomited several times.

June 8.—Urine was drawn off twice—morning and evening—and was observed to contain much blood. During day he vomited four times. He was ordered gallic acid and ice.

June 9.—Patient much weaker and urine drawn off by catheter is almost black from amount of contained blood. About 8 P.M. he passed into a state of collapse, which continued until death at 3 A.M. on June 10.

Autopsy.—No evidence of peritonitis. Bladder lay collapsed and flaccid in front of intestines, filling up nearly all front part of abdomen, and its apex being within 1½ in. of ensiform cartilage. The reflexion of peritoneum from bladder to abdominal wall was within 1 in. of umbilicus. A small red spot, like a flea-bite, near fundus, corresponded to puncture. Bladder contained a considerable quantity of bloody urine; muscular coat thickened; mucous membrane thrown into prominent folds, which were very red and congested, spaces between being pale. Ureters dilated and kidneys sacculated: in left kidney very little secreting tissue remained. Prostate enormously enlarged, almost filling pelvic cavity.

The next case occurred in my own practice. It is similar to the last, but the patient was younger and made a good recovery, a result, I believe, rather unusual under such circumstances.

CASE CL.—*Distended Urinary Bladder mistaken for Ascites.*

On March 3, 1876, Mr. Samuel L——, aged 46, was led into my consulting room. He was in great suffering, and I was informed that two homœopathic doctors, one a consultant of some eminence, had stated that he was suffering from disease of liver and dropsy, and that his care was hopeless. On examination, I found abdomen greatly distended, girth at umbilicus being 38½ in.; from ensiform cartilage to navel 9 in. and from navel to pubes 5½ in. The swelling was evidently due to fluid, which was encysted, lying in front of bowels, and rising from pelvis to within an inch of sternum. Both flanks tympanitic over an equal extent and cyst was central. Patient stated that for many years he had had a stricture of urethra, which had given him no trouble, and that he had enjoyed good health until about twelve months before, when abdomen began to swell slowly. Three weeks before, he had taken a cold bath, and since then he had had to pass water every hour and had some difficulty in voiding it. Altogether, however, he passed a fair quantity, and its characters were normal. No attempt had been made to pass a catheter. No symptom of disease of liver.

I advised that patient should see a surgeon, and have a catheter passed into bladder. This was not done, and on night of March 4 I was sent for in haste to see patient, as he was believed to be dying. At my suggestion, Mr. Berkeley Hill went to see him, provided with a suitable instrument. He met with some obstruction at neck of bladder, but succeeded in drawing off 7½ pints or 150 oz. of clear healthy urine, and in producing an immediate subsidence of the abdominal tumour. It was thought better not to empty bladder, but a bandage was applied round abdomen, and next morning 5 pints more were drawn off.

Patient had no bad symptoms; a catheter was passed regularly twice a day; on March 15 he was going on well and had returned to business, and in October he was in the enjoyment of good health, and there had been no return of swelling.

In the next case the peritoneum contained fluid as the result of acute peritonitis.

CASE CLI.—*Fluid in Peritoneum from Acute Peritonitis due to a Kick over a Congenital Hernia.*

Herbert R——, aged 12, admitted into Middlesex Hosp. October 28, 1866. Since four years of age he had been known to have an inguinal hernia on right side, but this had never caused him much inconvenience: and, excepting infectious diseases of childhood, he had enjoyed good health until two days before admission. On morning of Oct. 26 he received a kick while in bed over right testicle. This was followed by considerable pain in testicle, but he got up and went to school. In afternoon he had a rigor lasting for half an hour, followed by diarrhœa, and later in evening by vomiting and pain at epigastrium, extending thence over whole abdomen. During whole of 27th and following night he had frequent rigors and vomiting and urgent diarrhœa with light-coloured stools.

On admission, pulse 144; temperature 104°; respirations 34 and thoracic; abdomen distended and extremely tender, especially in right groin; right testicle much enlarged and exquisitely tender; cardiac and pulmonary signs normal. Boy was deaf and confused in mind, like a patient suffering from fever. Poultices with laudanum were applied to abdomen, and within first 24 hours after admission patient took as much as 5 gr. of opium internally. Under this treatment acute symptoms subsided, pain and vomiting diminished and diarrhœa ceased; but no material improvement took place. Tongue became dry and brown; prostration increased; cheeks were sunken and features pinched; and there was occasional delirium.

On Oct. 30 temperature was normal (98°), but pulse 132; abdomen more distended, and distinct evidence on tapping of fluid in peritoneum. Bowels confined, but vomiting had returned. On Nov. 1 temperature still 98°, but no improvement in patient's general condition.

The diagnosis in this case, as frequently stated at bedside, was that patient was suffering from acute peritonitis excited by kick on scrotum, inflammation having been propagated to peritoneum from hernial sac. It was suggested, however, by a gentleman who accompanied me on my visits, that possibly a portion of bowel had become strangulated in neck of hernial sac; but although this view was favoured by fact that bowels had not acted since boy's admission, the bowels had been very relaxed previously and the constipation was accounted for by the opium that had been taken subsequently. One of my surgical colleagues, however, who at my request saw patient in my absence, thought that probably there was a portion of bowel or omentum strangulated in sac and cut down upon it. A little pus escaped, but none of intestinal contents were found in sac. The boy gradually sank, and died at 8 A.M. on following morning.

Autopsy.—Chief morbid appearance was very extensive recent peritonitis, surface of liver and whole of bowels being plastered over with soft yellow lymph. Peritoneum also contained two or three pints of purulent fluid. No ulceration, perforation, nor gangrene of any portion of stomach, bowel, or appendix vermiformis, to account for peritonitis. No bowel nor omentum in hernial sac, nor even adherent in neighbourhood of internal opening. Intense vascular injection of outer surface of right testicle.

In the following case fluid was thrown out into the abdomen as the result of sub-acute peritonitis. But the chief pathological interest of the case lay in the fact, that the appearances found in the liver after death corresponded in every way with those which have been so often described of late years as constituting one of the lesions of constitutional syphilis (see pp. 141, 276), and yet that the evidence was as strong as negative evidence can well be in such a matter, against the view that the patient had ever suffered from syphilis.

CASE CLII.—*Fluid in Peritoneum from Chronic Peritonitis—Chronic Atrophy of Liver with Fibroid (probably syphilitic) Nodules in its interior.*

P. D——, aged 67, admitted into Middlesex Hosp. Jan. 17, 1867. He was a labourer, had been married for 23 years, and was the father of seven children. His oldest child was 22; all were in good health, and none had died; his wife had never had any miscarriages. A brother had died of consumption, but patient himself had always enjoyed good health, except for about three months three or four years previously, during which time he had been in hospital for a fracture of arm and other injuries. He had never had rheumatic fever, dropsy, jaundice, vomiting, nor hæmorrhoids, and denied syphilis. His habits had been temperate. Six weeks before admission a heavy weight fell upon his head, which wounded scalp and stunned him for a few seconds. For four days after this he gave up his work, and complained of pain in region of liver. On fifth day he returned to work, but after a few hours he was obliged to give it up. A week after accident he noticed that abdomen was swelling, and a fortnight after he began to vomit his food as soon as it was swallowed. Between this date and that of admission he had become very emaciated.

At time of admission patient was greatly emaciated, and countenance was expressive of suffering. Tongue moist and coated with a yellowish fur; no appetite, but patient complained of thirst, although he was afraid to drink, as everything he took was rejected within a quarter of an hour. He distinctly stated, however, that he had no pain between swallowing and vomiting. Abdomen considerably distended, girth at umbilicus measuring 33½ in.; unmistakable evidence of fluid in peritoneum. Hepatic dulness in right mammary line measured only 3 in.; no jaundice. Splenic dulness not increased; no enlargement of subcutaneous veins of abdomen. Immediately above and below umbilicus an obscure induration, with no defined margin, and, at some parts, yielding clear percussion. Abdomen generally tender, but by no means acutely so; fair movement of abdominal muscles in respiration. Patient stated that he was not in much pain, but he seemed to be always easier when lying on back with legs drawn up. Bowels regularly open. Pulse 96 and regular; cardiac dulness less than natural; sounds normal. Respirations 26; coarse crepitus over bases of both lungs, without any dulness or tubular breathing. Not the slightest œdema of legs, trunk, nor face, and urine free from albumen.

All efforts to relieve vomiting proved unavailing. Size of abdomen remained stationary. On Jan. 20 hiccough came on, and, notwithstanding frequent exhibition of nutritious enemata, emaciation rapidly increased until Jan. 28, when patient died from exhaustion.

At *autopsy*, one gallon of turbid fluid in abdominal cavity in front of small intestines, which were firmly matted together, forming a globular mass pointing towards umbilicus and accounting for obscure tumour felt during life. Peritoneum was everywhere coated with a thick layer of reticulated lymph and great omentum much thickened and indurated, but nowhere about bowels nor mesenteric glands was any indication of tubercular or cancerous deposit. Stomach much contracted, and mucous membrane for several inches from pylorus reddened and thrown into folds and under microscope presented a remarkably villous appearance; pyloric end surrounded and pressed on by greatly thickened omentum, but nowhere in its coats could any appearance or structure resembling that of cancer be discovered. Liver small, weighing only 40 oz.; capsule at some places thickened and adherent to surrounding parts, and over surface were several cicatrix-like depressions. Scattered through substance of liver were numerous rounded opaque-yellow deposits, largest about size of a cherry. Such of deposits as were immediately beneath capsule were not at all raised above general surface, while others were situated at bottom of cicatrix-like depressions. On section they presented a firm, fibrous-looking appearance, and yielded no milky juice. On microscopic examination they were found to be made up of white fibrous tissue, with nuclei and small fibre-cells and granular matter, but to contain nothing suggestive of cancer. Spleen small; lungs congested and œdematous at bases; kidneys slightly granular; heart small, but healthy. No deposits like those in liver could be found in any other organ, and no cicatrices could be discovered on penis, in groins, or on legs.

The three following cases were good examples of fluid in the peritoneum due to cancerous peritonitis.

CASE CLIII.—*Primary Cancer of Peritoneum, causing a large Effusion of Fluid.*

Jane A——, aged 51, nurse in a private family, was admitted into King's College Hospital under my care on July 22, 1859. Her general health had always been good prior to illness for which she was admitted. She had never had any other illness of importance, and had always lived comfortably and been temperate in her habits. No hereditary tendency to cancer could be ascertained. Three months before admission she began to complain of pain in lower part of belly, in situation of left ovary and of bladder, and also in back. The pain above pubes was always worse after micturition. She was thought to have inflammation of bladder; leeches were applied and pain abated. Still she kept at work until a fortnight before admission, when she was seized somewhat suddenly with febrile symptoms, vomiting, much pain and tenderness of abdomen, followed by swelling, which rapidly increased.

On admission, patient emaciated, but abdomen greatly distended, measuring 36½ in. at umbilicus, and presenting all the characters of fluid in peritoneum with addition of great tenderness on pressure, especially on left side. Pulse 92; tongue very red and clean; bowels constipated, but easily acted on by medicine; and occasionally much vomiting. No enlargement of liver or spleen, and no jaundice; no anasarca of legs; heart's sounds normal; urine scanty and dark, but contained no albumen.

Treatment consisted in effervescing draughts with hydrocyanic acid to allay sickness, a diuretic pill containing squill, digitalis, and blue-pill, and colocynth and henbane pills to keep bowels open. At first diuretics increased flow of urine, and size of abdomen remained stationary; but about middle of August they seemed to lose their effect, abdomen became larger, while tympanitic portion, which always was uppermost in whatever position patient lay, became smaller. On Aug. 27 abdomen measured 40 in. in circumference, and patient complained much of its feeling very tight and painful; respirations 38 and thoracic, and much dyspnœa from pressure of fluid on diaphragm; nothing abnormal in physical signs of lungs. Tongue still very red and clean; vomiting more frequent and urgent; bowels never opened without medicine. From first she had continued losing flesh, and her features were now pinched, and she slept little. To-day abdomen was tapped, and about two gallons of a transparent greenish-yellow fluid, having a specific gravity of 1020 and containing much albumen and also many white flakes of fibrillated lymph, were drawn off. For two days afterwards patient experienced great relief; sickness and pain ceased. No tumour could be felt in abdomen after evacuation of fluid. On morning of Aug. 30 patient was seized with severe vomiting and a return of pain and tenderness in abdomen, which was much distended and tympanitic, while at same time there was evidence of a small quantity of fluid in peritoneum. Vomiting, emaciation, and abdominal pain continued, uninfluenced by treatment. Patient could bear nothing in her stomach except champagne and ice, and for four weeks she was nourished by enemata of beef-tea, eggs, and brandy, with a few drops of laudanum. Aphthæ appeared on tongue. Fluid did not reaccumulate in large quantity in abdomen, but early in September several small nodules could be felt through abdominal parietes. On Sept. 18 left thigh and leg were noted to be swollen, and there was tenderness along course of femoral vein. After this patient was thought on several occasions to be moribund; but she rallied, to die at last on Sept. 28, in a state of extreme emaciation.

Autopsy.—Only 30 oz. of clear straw-coloured fluid were found in the peritoneum. The peritoneal surface of all the intestines, of the liver, and of the bladder, were studded over with innumerable nodules of cancer, varying in size from a pin's head to a hazel-nut. The mesenteric glands were also slightly enlarged from cancerous deposit. The cancerous masses exuded a milky juice on section, which contained characteristic 'cancer-cells.' The intestines were connected here and there by a few loose adhesions, and the sigmoid flexure was f bound down over the iliac vein by firmer adhesions and nod cancer. There was no cancer of the mucous or muscular coat stomach or bowels, or in the uterus, liver, kidneys, or lung apex of the right lung were several cretaceous nodules. The liver and spleen were of normal size. The left iliac and femoral veins were plugged by adherent coagula.

CASE CLIV.—*Cancerous Peritonitis and Tumour of Omentum.*

Catherine H——, 38, charwoman, adm. into Middlesex Hosp. Jan. 16, 1869. Father died at 78 of 'old age'; mother (40) and two sisters (39 and 40) died of consumption; one sister alive and well. No history of cancer in family. Excepting occasional cough, patient had enjoyed good health till 12 weeks before admission, when she began to complain of fulness and tension of stomach after eating, and after four weeks she was attacked with shivering, severe lancinating pain in abdomen and back, and occasional vomiting. A month before admission vomiting became more urgent and abdomen began to swell, and swelling increased so rapidly that paracentesis had been proposed to relieve dyspnœa. With these symptoms were constipation, scanty urine, and emaciation, but no night-sweats. A tumour had also been detected between umbilicus and pubes.

State on admission.—Emaciated, with anxious expression, but no jaundice nor dilatation of capillaries of face. Complains of severe pain and swelling of abdomen, which measures at umbilicus 33½ in. and presents distinct signs of fluctuation, and at same time is extremely tender, so that slightest tapping causes intense suffering. The pain is constant, but subject to exacerbations. Hepatic dulness does not ascend too high. No enlargement of abdominal veins nor of spleen. Between umbilicus and pubes is a distinct central hard tumour, and on vaginal examination Dr. Hall Davis reports, 'uterus generally enlarged, probably from carcinomatous disease.' Tongue moist, slightly furred, rather too red; frequent retching; bowels costive. Pulse 108, feeble; no abnormal sound over heart. Respirations 60, entirely thoracic; lungs healthy. Urine 1024; no albumen.

Patient was ordered a grain of opium every eight hours and laudanum fomentations to abdomen. By this treatment pain and vomiting were for a time relieved, but on Jan. 21 pain again so severe that it was necessary to have recourse to subcutaneous injections of morphia, and by Feb. 11 as much as one grain of morphia was injected three times a day. On Jan. 25 slight pitting of legs; on 28th, tongue dry, red, and smooth, with aphthous patches. On Feb. 19 patient was seized with acute pain in left side of chest, over which pleuritic friction and crepitation audible. Abdominal swelling did not increase much, but exhaustion and emaciation were daily more marked, until at length patient sank and died on Feb. 27.

Autopsy.—Abdomen only examined. Peritoneum contained several pints of bloody serum. Inner surface of abdominal wall was coated with a rough layer of recent lymph, and intestines and mesentery studded with numerous small nodules of cancer, some larger than a pea. Tumour felt during life was not uterus, but a mass of cancerous deposit in omentum.

CASE CLV.—*Cancerous Peritonitis.—Tumour of Omentum—Double Pleurisy.*

Mary Ann P——, 48, adm. into Middlesex Hosp. March 9, 1871. Father (47), one brother, one sister, and two of her own children had died of consumption. Mother died at 46; for three months before death had jaundice and enlargement of liver (cancer?) Married and had seven children and one miscarriage; catamenia ceased two years ago. For several years had a winter-cough, and during last 12 months had lost flesh and often perspired profusely at night Eleven weeks before admission became very weak and lost appetite, and after four or five days began to have sour eructations after food as well as at other times. Six weeks before admission began to suffer from pain in epigastrium and left side of abdomen and about right shoulder-blade. Two weeks later abdomen began to swell, and a fortnight before admission swelling was first observed in legs. Bowels had been costive; night-sweats had ceased when abdomen began to swell.

State on admission.—Very weak and emaciated; slight œdema of legs. Complains of pain and swelling of abdomen, which measures at umbilicus 34 in. Has all signs of fluid in peritoneum. In left side of abdomen a hard swelling also felt extending from under left ribs downwards and forwards to 2 in. below level of umbilicus, smooth, with no sign of splenic notch, and not tender. Liver cannot be felt. Abdominal veins enlarged. Pain commences at epigastrium, and stretches down to left side and up to right shoulder; it often comes on suddenly and severely, quite irrespectively of taking food, and lasts for about 20 minutes. No jaundice. Tongue clean; appetite bad; often vomits food within half an hour, and most solid food causes pain until it is rejected. Pulse 84; sounds of heart healthy. Frequent loose cough with mucous expectoration; considerable dyspnœa; mucous râles heard over both lungs, most abundant on left side. Temp. 101·5°. Sleeps fairly. Urine 1019; alkaline; no albumen, no bile-pigment and no lithates.

Patient was ordered a mixture of quinine, iron, and spirit of nitrous ether, with wine. At first she improved; vomiting ceased; pain was relieved; she was able to eat and retain meat; and abdomen gradually diminished until on March 24 girth of abdomen was only 29 in.; no sign of fluid in peritoneum could be detected and there was no œdema of legs. Splenic dulness could now be separated by a tympanitic space 1½ in. in breadth from tumour, and finger could be inserted between upper border of this and lower left ribs. No bowel could be made out in front of tumour, and there was tympanitic percussion note behind it. On vaginal examination tumour did not appear to have any connection with uterus or ovaries. Notwithstanding these signs of improvement, attacks of pain became more severe, so that on March 31 it was necessary for first time to have recourse to morphia, and on April 12 abdomen was covered with belladonna and glycerine. On April 26 there was again fluid in peritoneum, and abdomen measured 32 in.; and on April 28 patient vomited for first time since admission. On May 1, increased dyspnœa with lividity of lips, and over lower third of both lungs dulness with feeble breathing. Œdema of legs had also returned. No pyrexia. A pill of digitalis, squill, blue pill, and morphia twice daily was now ordered. On May 5 abdomen measured 33 in., and there was dulness with feeble breathing over lower half of both lungs. Vomiting now became frequent, and on May 15 diarrhœa (4 stools daily) set in. Abdomen again became reduced until on May 29 girth was only 29 in., but patient became rapidly weaker and died on May 31.

Autopsy.—Four pints of turbid flaky serum in peritoneum. Loose bands of adhesion passed from abdominal parietes through fluid to intestines, which were bound by adhesions into a rounded mass. Peritoneal coat of bowels intensely congested and studded with numerous opaque white deposits of cancer. Tumour felt during life in left side of abdomen consisted of omentum greatly thickened by cancerous infiltration and fibrous tissue, and lying in front of left kidney and descending colon. Both ovaries about size of small oranges, and made up of soft cancerous matter with cysts containing a gelatinous fluid. Liver small, rounded, and firm; its capsule greatly thickened, at some places to extent of 2 or 3 lines. Kidneys healthy. Heart healthy. Right pleura contained 22 oz., and left, 44 oz. of turbid serum. Both lungs condensed and carnified, and outer surface of both coated with rough membrane or lymph, which could be readily peeled off. No deposit in either.

In the two next cases there was a considerable accumulation of fluid in the peritoneum due to tubercular peritonitis. Case CLVI. resembled two of the cases of cancerous peritonitis in the fact that there was an omental tumour; while Case CLVII. was remarkable in the disappearance of the fluid and the recovery of the patient under treatment.

CASE CLVI.—*Fluid in Peritoneum from Tubercular Peritonitis—Tubercle of Omentum and Paracentesis—Death.*

Charles A——, 28, India-rubber worker, adm. into St. Thomas's Hosp., May 25, 1876. Father, mother, and five brothers and sisters alive and healthy; 4 brothers and sisters died in infancy; no phthisis in family. Excepting 'bilious attacks,' had good health till two years ago; since then had drunk a good deal of beer and spirits, and had complained of palpitations and dyspnœa. Six weeks ago coughed up a little blood on several occasions. Three weeks ago began to have pain across upper part of abdomen, with nausea, anorexia, and occasional vomiting of viscid mucus, especially in morning. Bowels also became relaxed; 6 or 8 dark brown liquid motions in day. Worked till day before admission.

On admission, pale and thin; no jaundice nor stellate veins on cheeks. Abdomen much distended, measuring 35 in. at umbilicus. Great tympanitic prominence between umbilicus and ensiform cartilage; also considerable ascites. Upper margin of hepatic dulness rose to within half an inch of nipple; lower margin could not be made out. Spleen not large. No appreciable tumour. No tenderness of abdomen, but considerable pain at times across upper part. Diarrhœa persisted, but vomiting had ceased. No cough; no abnormal signs in lungs. P. 108; sounds of heart normal. Temp. at night rose to 102° or 103°. Urine 1020, high-coloured; no albumen.

Patient was treated with diuretics and a nutritious diet, and subsequently with quinine and mineral acids. Diarrhœa persisted, and fluid in abdomen increased. On June 7 girth 37½ in., much pain in abdomen and some dyspnœa. Nine pints of fluid were drawn off by paracentesis; this was of a greenish colour, alkaline and clear, but contained flakes of fibrillated lymph entangling blood-cells; sp. gr. 1020. After this, breathing was relieved and fluid did not collect again to any extent. But he continued to get thinner, and about June 12 he complained much of cough, and bronchitic râles were heard over both lungs. During June he had occasional hectic flush on cheeks; evening temp. varied from 99·4° to 101·5°; no decided night-sweats. On July 1 girth at umbilicus only 31 in.; an obscure induration could be felt, apparently in the omentum, stretching from umbilicus to left ribs; its position not affected by deep inspiration. After this, emaciation rapidly increased, and indurated mass could be felt extending across upper part of abdomen to right ribs; cough became more frequent, and there was expectoration of viscid mucus which gradually became puriform. Diarrhœa persisted and vomiting returned; but at no time were there night-sweats. Death by exhaustion on July 31.

Autopsy.—Peritoneum contained six pints of clear yellow serum, part of which was contained in separate cavities bounded by lymph. Great omentum everywhere infiltrated with a thick firm tubercular mass, adherent to the parietal peritoneum and to the intestines. Serous covering of bowels studded with small nodules of tubercle, and glued by soft lymph to one another and to surrounding parts; two small tubercular ulcers in ileum. Liver somewhat enlarged and fatty. Small deposits of tubercle becoming caseous in centre scattered through both lungs, and in upper part of left lung larger masses of soft caseous tubercle breaking down into cavity. Apex of left lung firmly adherent and marked by cicatricial depressions; 16 oz. of serum in left pleura, and 4 oz. in right. Heart and other organs healthy.

CASE CLVII.—*Fluid in Peritoneum from Tubercular Peritonitis. Recovery under treatment.*

On Feb. 18, 1876, I saw in consultation with Mr. A. Maclaren, Miss M——, aged 21. She had always been delicate, and in previous autumn had been laid up four or five weeks with pleurisy on left side. About four weeks before I saw her, first noticed her abdomen enlarging, with some pain but not at all severe, and since then she had been getting thinner and weaker and she had perspired during sleep. On examination abdomen was tense and evidently contained a small quantity of fluid, but was not tender. Girth at umbilicus 32 in. No sign of tumour. Tongue coated; appetite fair; no nausea nor retching; bowels confined. Pulse 108. Temp. 101° to 103°. No cough; but dulness, flattening, and crepitation below left clavicle. Urine scanty and dark. Patient was ordered quinine and iron in conjunction with digitalis. On Feb. 23 already felt better; urine more copious; abdomen less tense; girth 31 in.; pulse 84; temp. 99° to 102·5°. Feb. 28: Girth 29½ in.; pulse 60 and irregular; temp. 98·6°–101·5° Still feels better; to continue iron and quinine, but to omit digitalis. The digitalis was resumed from time to time, and patient also took iodide of potassium and syrup of iodide of iron and cod liver oil. On March 13, girth 28½ in.; pulse 64; temp. had not exceeded 100° at any time during a week. On March 20, girth 27 in., no sign of fluid in abdomen; intestines feel matted together; temp. 99°. April 10: There has been no rise of temperature, and continues to improve; intestines still feel matted; still dulness at left apex, but no moist sounds. After this patient continued to improve, and remained well all summer; but in October I heard that she was again laid up with fever and signs of mischief in right lung.

Case CLVIII. appeared to be one of those already referred to (p. 444), where a quantity of fluid collects in the peritoneum, as the result of a subacute inflammatory process.

CASE CLVIII.—*Ascites—Anæmia.* (*Tubercle?*)

Martha W——, aged 11, adm. into Middlesex Hosp. July 17, 1868. Father died of phthisis; mother living and healthy; one sister living and healthy; no brothers. Some years before had measles and whooping cough. Last spring had a cough and expectoration for some months, but no night-sweats. Cough got better, but about a month before admission abdomen began to swell, followed after three days by rather severe diarrhœa and loss of flesh. Eight days before admission patient was tapped 1½ in. below the umbilicus, and 8 pints of clear transparent fluid drawn off. Three or four days after tapping diarrhœa subsided.

On admission, child was thin and anæmic; abdomen distended with fluid, measuring 24¾ in. at umbilicus. No œdema; no evidence of disease of heart or kidneys; no enlargement nor pain of liver. Pulse 120; no pyrexia nor night-sweats. Tongue clean; appetite good; bowels quiet; some prolapsus ani.

A good diet and perchloride of iron with spirit of nitrous ether were prescribed. Under this treatment ascites slowly diminished, and by Aug. 15 all trace of it had disappeared; girth of umbilicus was only 21¼ in., and general health seemed good.

The next case was a remarkable one. Notwithstanding the unusual degree of tenderness, the slow pulse, the low temperature, and the paroxysmal character of the pain led us to regard the case, in the first instance, as merely one of severe colic. Its pathology, however, was probably similar to that of the last case.

CASE CLIX.—*Symptoms of Colic followed by signs of Fluid in Peritoneum.*

Edward J——, aged 21, who had formerly been a printer, but had been working for six weeks at a carver and gilder's, was admitted into Middlesex Hospital April 12, 1868. On April 6 he had been suddenly seized with severe pain in abdomen and retching. Pain had been constant ever since, but had been liable to severe exacerbations; vomiting had recurred daily, but had not been so violent as at first. Bowels had acted on 8th and 10th after castor oil and laudanum. Shortly before attack patient had been suffering from gonorrhœa, and he stated that some years before he had a similar, though much less severe, attack of abdominal pain.

On admission patient still complained of constant pain in abdomen, with frequent acute exacerbations; pain was increased by any movement, and there was also considerable tenderness over abdomen, most marked over the cæcum. Abdomen distended and tympanitic, and breathing entirely thoracic; frequent retching of scanty bilious matter. A dark red (not blue) line along margin of gums; tongue moist and only slightly furred; thirst; bowels had not been open for two days. Pulse 84; skin cool; temperature under tongue 97°; no albumen in urine.

Patient was ordered a warm bath, warm fomentations to belly, an enema of three pints of barley-water with four drachms of tincture of assafœtida, and a grain of opium every four hours. Enema brought away two copious motions, but with no relief to pain. On April 13 a third of a grain of extract of belladonna was ordered every three hours, but next day pain, tenderness, and tension of abdomen had increased, although pulse was only 72, and temperature 97°. He was again ordered a grain of opium every four hours, a draught of castor oil and laudanum, and frequent enemata. He continued taking six grains of opium a day until April 17, and then three grains until April 23. Under this treatment bowels were freely moved, and paroxysms of pain less severe; but he still had occasional vomiting, abdomen grew larger and more tense, and on April 19 there was unmistakable evidence of fluid in peritoneum. A thrill could be propagated from one side to the other on tapping, and when patient was supine there was dulness in either flank, which varied with his posture. He still had occasional paroxysms of pain, but no tenderness of the abdomen. Pulse, however, kept steadily at 72, and temperature rarely exceeded 98°. Signs of fluid in peritoneum with occasional slight paroxysms of pain continued until May 4. After this abdomen gradually became smaller, and on May 18 it had regained its normal size and presented no sign of fluid, and patient left hospital free from pain.

Case CLX. was a good illustration of fluid in the peritoneum resulting from disease of the kidneys. During life the case was regarded as an example of the large white kidney following nephritis and passing into the fatty kidney; and although one of the kidneys was unexpectedly found very contracted after death from some old disease, you will observe that the other has *three* times the size and weight of a normal kidney.

CASE CLX.—*Fluid in Peritoneum from disease of Kidney—Albuminuria and General Anasarca—Pericarditis and Pleurisy—Death by Uræmia—Great Hypertrophy of Left Kidney and Atrophy of Right.*

James S——, aged 23, was admitted into Middlesex Hospital March 12, 1868. Excepting an attack of 'gastric fever' 2½ years before, his previous health had been good. He had never suffered from scarlet fever; but for nine years he had been in habit of working in a very hot room, and of drinking gin as well as beer daily. Six weeks before admission he went from the West End to the City one evening and got wet through. Three nights after this, on taking off his boots he noticed that feet were swollen, and next morning there was slight swelling of legs, thighs, trunk, and even of face. He continued working for two or three days and then went to St. Bartholomew's Hospital, where he remained a month, but got worse rather than better. Four or five days before coming to hospital he found his breath becoming short.

On admission the patient's countenance extremely anæmic, pasty and puffy; considerable œdematous swelling of trunk, extremities, and which was scanty, but contained no albumen. Mind clear; slept very badly, owing to orthopnœa.

Patient was treated with diuretics (nitrous ether, acetate of potash, and decoction of broom-tops, with a pill of digitalis, blue pill and squill), and stimulants (gin 8 oz. and brandy 4 oz.), while mustard and linseed-poultices were applied over chest. Prostration and dyspnœa nevertheless increased. On Feb. 26 pulse 86, extremely feeble and irregular; respirations 48, but interrupted by frequent cough; expectoration consisted of nummular masses of yellow pus; veins of neck turgid, with slight regurgitation from below. Bowels had acted six or seven times in night. Dropsy had increased, and right hand and arm now œdematous. These symptoms continued until patient's death at midnight between Feb. 27 and 28.

Autopsy.—Heart weighed 14 oz.; mitral orifice much constricted, just admitting tip of finger, and flaps of valve very rigid and thickened from fibrous and calcareous deposit; also calcareous deposit in aortic valves, which, however, were competent; tricuspid orifice slightly dilated. Firm adhesions between opposed surfaces of both pleuræ; pulmonary pleuræ thickened; and lower part of both lungs condensed from intersecting fibrous bands, with œdema and congestion of intervening pulmonary tissue; bronchial tubes dilated, walls thickened, and interior filled with pus. Peritoneum contained several quarts of yellow serum. Liver very small and dense, with much fibrous deposit on its surface and also extending into its interior; outer surface at several places granular, the depressions corresponding to centre of lobules; liver weighed only 31 oz. Mucous membrane of stomach intensely injected, with much adherent mucus and numerous hæmorrhagic erosions. Kidneys slightly granular.

Although in the following case the patient on three different occasions recovered, so that fortunately no opportunity was afforded for verifying our diagnosis, there was little doubt that the fluid in the peritoneum was, as in several cases which I brought under your notice in former lectures (Lect. IV. and VIII.), due to portal obstruction from cirrhosis of the liver.

CASE CLXII.—*History of Spirit-drinking—Cirrhosis of Liver—Enlarged Spleen—Ascites—Gastro-enteritis—Epistaxis and Hæmatemesis—Removal of Ascites by Diuretics and other remedies on three different occasions.*

Etienne D——, aged 39, a pipe-maker, was admitted into Middlesex Hosp. under my care on April 23, 1868. His mother was alive, aged 76; but his father at 61, and a brother at 28, had died from effects of hard drinking. Patient himself had for many years been in habit of drinking largely of beer and spirits, especially the latter, and for last three years he had been very intemperate, taking large quantities of brandy and rum.

Although he had never had an attack of delirium tremens, he had often been very shaky in morning. Notwithstanding his habits, he had enjoyed good health until three years before admission, when he began to suffer about every week or ten days from severe pinching pains in abdomen followed by diarrhœa, which was checked by chalk-mixture. Nine months before admission he began to retch in morning three or four times a week, and twelve months before admission he had an attack of bleeding piles. During last year also gums had been liable to bleed, and he had been losing flesh. Five weeks before admission vomiting became more urgent, and he began to suffer from attacks of acute twisting pain in right side of abdomen. A fortnight later vomiting subsided, but diarrhœa came on, and he first noticed abdomen begin to swell; this swelling rapidly increased and during last week had caused much dyspnœa. He continued, however, drinking port wine and brandy and water until admission.

State on admission was as follows:—Body spare, with sharp features; face sallow; distinct icteric tint of conjunctivæ, but no evident jaundice of trunk or extremities; capillaries of face much enlarged; no puffiness of face nor anasarca of extremities. Chief complaints are of great weakness and of swelling and pain in abdomen, which is not tender, but is much distended and measures 34 in. at umbilicus. Enlargement is evidently due to fluid in peritoneum. Abdominal veins unusually distinct and slightly enlarged, especially in cæcal region, where they form a distinct network connected with the veins ascending to chest. Hepatic dulness in r. m. l. cannot be made out distinctly. In epigastrium liver can be felt through abdominal parietes; it is hard and resisting, without any appreciable nodulation. Splenic dulness increased, measuring vertically 4 in. and extending forwards to within 2½ in. of line of nipple. Tongue coated with a thick yellow fur; appetite indifferent; bowels open about four times a day; motions watery and yellow. Pulse 108; physical signs of heart and lungs normal. Urine acid; sp. gr. 1017; free from albumen, but contains a small quantity of bile-pigment.

Patient was ordered a draught four times a day containing acetate of potash (gr. xx), spirit of nitrous ether (ʒss), and decoction of broom-tops (℥jss); and a pill twice a day with squill (gr. jss), powder of digitalis (gr. ss), and blue pill (gr. iij). An ointment composed of equal parts of blue ointment and ung. belladonnæ was applied over abdomen. Alcohol in every shape was interdicted, and diet was restricted to milk, beef-tea, and farinaceous articles. No attempt was made to check diarrhœa. He at once began to improve. He had frequently colicky pains in abdomen, and on May 2, 3, 6, and 7 he had attacks of vomiting, and on May 7 slight epistaxis. But from first, flow of urine increased and ascites diminished, as shown by following measurements:—

Girth at umbilicus, April 23, 34 inches.
" 27, 33·6 "
" May 4, 31·75 "
" 6, 30·75 "
" 11, 29·25 "
" 22, 28·25 "

On May 22 patient was greatly better. Abdomen of natural size; no evidence of ascites. Purging and vomiting had ceased; the appetite improved, and he could eat and retain meat. Splenic dulness was reduced, and hepatic dulness in right mammary line measured $3\frac{1}{2}$ in., apparent increase in latter being probably due to its lower margin being no longer obscured by bowels distended with gas. In epigastrium liver could be felt hard and obscurely nodulated.

On June 22 he left hospital, without any return of dropsy and gaining flesh.

After leaving hospital he gradually returned to his former habits of drinking largely of beer and spirits. But with exception of occasional epistaxis and an attack of diarrhœa in summer of 1869, he had fair health until beginning of March 1870, when he began to suffer from great nausea, retching in morning, pain and tenderness in hepatic region, and swelling of abdomen. About end of May legs began to swell, breathing became embarrassed, and once he vomited about a pint of blood. On June 30 he was again admitted into Middlesex Hospital under my care.

Chief symptoms were these:—Face sallow, much itching of skin, but no jaundice; frequent retching, but does not vomit food, and appetite good; tongue unusually smooth, devoid of papillæ and red; 2 loose motions in day. Girth at umbilicus $34\frac{1}{2}$ in.; considerable ascites; liver large and firm, but smooth; hepatic dulness in r. m. l. $5\frac{3}{4}$ in. and lower edge $2\frac{1}{4}$ in. below ribs; no obvious enlargement of spleen. Much tense œdema of legs. Urine clear and containing a trace of bile-pigment, but no albumen. Pulse 76; heart-sounds normal. Respiration embarrassed, but no signs of œdema of lungs, nor of fluid in pleura. Bismuth and a milk diet were first given to allay sickness, and afterwards treatment consisted in pills of blue pill, squill and digitalis, a mixture of perchloride of iron and nitrous ether, and occasional saline aperients. He again speedily improved, and on July 28 he left hospital, with a good appetite, no retching and no appreciable ascites, girth at umbilicus being 32 in., but no diminution in size of liver.

On March 12, 1873, patient was admitted a third time into Middlesex Hosp. under Dr. H. Thomson with similar symptoms to those of his two former attacks, but with addition of general bronchitis and fibroid (?) consolidation of right lung. Liver now extended to an inch below umbilicus, and its surface was distinctly nodulated. He again left hospital on May 29, much better. Some months after leaving hospital, dropsy returned, with large sloughing sores on legs, under which patient sank. There was no post-mortem examination.

In a former lecture (Lect. VI.) I have called your attention to cases of ascites resulting from cancer of the liver. In the following case ascites without jaundice was produced by compression of the portal vein by a mass of cancerous glands in the portal fissure, just as we have found jaundice with or without ascites produced by a similar cause (Lect. X.).

CASE CLXIII.—*Cancer of Stomach and Omentum—Cancerous Glands compressing Portal Vein and causing Ascites—Cancerous Nodules in Abdominal Parietes.*

Charlotte H——, 54, adm. into Middlesex Hosp. March 5, 1869. Father lived to 84; mother and two grown-up sisters dead; cause unknown. Excepting a winter-cough for six years, patient had enjoyed good health until early in December 1868, when she began to suffer from pain in upper part of abdomen, vomiting after food, and great prostration, with loss of appetite and flesh. About same time she noticed abdomen and legs begin to swell.

State on admission.—Great prostration and vertigo; marked anæmia; moderate œdema of both legs. Abdomen greatly distended by fluid in peritoneum, but not tender; no appreciable tumour, and no obvious enlargement of liver, but beneath skin of abdominal parietes were several firm nodules, about size of peas. Tongue clean; appetite bad; bowels open; no vomiting, but much pain, after food. Pulse 108; arteries rigid; first sound of heart at base somewhat prolonged. Lungs healthy. Urine 1026; much lithates, but no albumen. Some numbness of left arm, and indistinctness of speech.

After admission, vomiting returned in an urgent form. Bismuth, creasote and opium, and blisters with morphia dressing failed to give relief. Vomited matters were dark green. Pain at epigastrium persisted. Brandy and beef-tea injections were administered, but patient rapidly sank and died on March 14.

Autopsy.—Six pints of clear serum in peritoneum. At pylorus was a large soft cancerous tumour extending 4 in. into stomach, its inner surface deeply excavated by ulceration, so as to form a cavity communicating above with stomach, and below with duodenum. Extensive cancerous deposit in glands of lesser omentum, one mass as large as a hen's egg compressing portal vein. Post-peritoneal glands in front of vertebral column also enlarged from cancer, forming a mass which pressed on vena cava. Under surface of diaphragm studded with small masses of cancer, and the small nodules felt during life in abdominal wall were of a similar structure. Much rigidity along attached margin of aortic valves, which were competent.

In the next case there was a large collection of fluid in the peritoneum due to compression of the portal vein by a colloid tumour. The fluid drawn off by paracentesis was turbid from the presence of blood and cells of colloid matter.

CASE CLXIV.—*Ascites—Colloid Disease of the Stomach and Peritoneum.*

Daniel G——, cabinet maker, aged 44, adm. into Middlesex Hosp. Dec. 1, 1868. No history of malignant disease in family. When 24 he had primary syphilis, followed ten years later by nodes on shins. Had been occasionally intemperate, but not a habitual tippler. Eight months before admission began to suffer from debility, loss of appetite, and constipation; and four months before, became jaundiced and continued so for a month. After this continued weak and low until nearly a month before admission, when he observed some swelling of belly, and after a few days he had an attack of acute pain in abdomen which lasted some hours. Swelling rapidly increased, and about a week before admission two gallons of clear straw-coloured serum were drawn off by paracentesis. This gave temporary relief, but fluid began at once to reaccumulate. Since swelling commenced he had taken much purgative medicine and had rapidly lost flesh.

On admission, no trace of jaundice nor of œdema of integuments, but abdomen enormously enlarged in comparison with very emaciated body, from presence of fluid in peritoneal cavity. Girth at umbilicus 35½ in. Veins of abdominal parietes dilated. No pain nor tenderness on pressure over abdomen. No appreciable tumour, but splenic dulness increased and hepatic dulness in r. m. l. rises as high as 6th rib, although lower edge of liver cannot be felt below ribs. Tongue unusually red and fissured in centre; gums sore and slightly swollen; frequent vomiting, and brings up food and medicine; bowels very relaxed—about 12 motions in 24 hours. Pulse 96, regular and feeble; respirations thoracic; organs in chest normal. Temp. 98·4°. Urine contains much lithates, but no albumen nor bile-pigment.

Remedies prescribed were ice to suck, with iron and digitalis, vegetable charcoal and stimulants. Diarrhœa abated, but swelling rapidly increased, and on Dec. 4, 7½ pints of fluid were drawn off by paracentesis. Fluid was turbid and reddish-brown; sp. gr. 1017; it contained no flakes of lymph, but under microscope presented numerous blood-corpuscles and several large cells with granular contents, exactly like those found in colloid material after death. After fluid was drawn off, still no tumour could be felt, and liver did not appear enlarged. Operation was followed by great, but temporary, relief. Fluid reaccumulated; vomiting persisted; and prostration increased until death by exhaustion on Dec. 8.

Autopsy.—Several pints of bloody serum in abdomen. An enormous deposit of colloid material in great omentum, forming a large mass covering intestines. Similar deposits on inner surface of abdominal parietes apposed to mass in omentum, on under surface of diaphragm and over liver. A large mass extending along lesser omentum into portal fissure surrounded and compressed portal vein, but interior of vessel free from coagulum. Growth nowhere penetrated into substance of liver. To naked eye, morbid material appeared to consist of an aggregation of small nodules of translucent gelatinous material, and it had the usual microscopic structure of colloid. At pyloric end of stomach and extending for 5 in. from valve was an extensive colloid deposit involving all the coats; mucous membrane over it had quite disappeared, leaving morbid tissue bare and exposed. Spleen invested by colloid deposit, but normal and weighed only 5½ oz.

LECTURE XIII.

A. *PAIN IN THE LIVER*—B. *GALL-STONES*—C. *ENLARGEMENTS OF GALL-BLADDER.*

A. HEPATIC PAIN SIMULATED BY: 1. PLEURODYNIA—2. INTERCOSTAL NEURALGIA—3. PLEURISY—4. GASTRIC DYSPEPSIA—5. INTESTINAL COLIC—6. RENAL COLIC. THE VARIETIES AND CAUSES OF GENUINE HEPATIC PAIN.

B. GALL-STONES. THEIR VARIOUS CONSEQUENCES AND SYMPTOMS.

C. ENLARGEMENT OF GALL-BLADDER. ITS CAUSES, CLINICAL CHARACTERS, AND TREATMENT.

A. HEPATIC PAIN.

Gentlemen,—Pain is sometimes the most prominent symptom of disease of the liver and is often an important aid to diagnosis. You must, however, beware of being misled by patients who constantly ascribe pain to the liver with which that organ is in no way concerned. In forming a diagnosis you must keep constantly in view the various conditions which may simulate hepatic pain. They are mainly:—1. Pleurodynia, 2. Intercostal Neuralgia, 3. Pleurisy, 4. Gastric Dyspepsia, 5. Intestinal Colic, 6. Renal Colic.

1. *Pleurodynia*, or rheumatism of the intercostal muscles, may be situated in the right hypochondrium, and then the acute pain, increased by pressure, by movement, by taking a long inspiration, or by coughing, and accompanied by short jerking respirations, may be mistaken for the pain of perihepatitis; but it differs from this—

a. In the pain being more localised. It is often confined to one spot between two ribs, and there is no tenderness on pressure over the epigastrium, or elsewhere in the hepatic region than in the spot to which the patient refers the pain.

b. In the absence of symptoms of pyrexia or of constitutional disturbance.

c. In the absence of any other symptom or sign of hepatic disease.

d. In the occasional coexistence of muscular rheumatism in other parts of the body.

2. *Intercostal Neuralgia,* except that the pain is more intermittent, may present many of the characters of pleurodynia, and may like it be localized in the hepatic region; but on the whole it is chiefly met with in the sixth to the ninth intercostal spaces on the left side, and in females. When it occurs in the hepatic region, it differs from true hepatic pain—

a. In the pain being chiefly referred to three points in the course of the nerve, viz. in the vertebral groove, in the axillary region, and at the termination of the nerve in front.

b. In the frequent coexistence of neuralgia of the mammary gland, tenderness over one of the dorsal spines, or cutaneous hyperæsthesia.

c. In the absence of any other symptom or sign of hepatic disease.

You must not forget, however, what I told you in a former lecture, that intercostal neuralgia may have a hepatic origin and may be a sequel of pain which is truly in the liver (p. 338).

3. *Pleurisy* may give rise to a pain, which, like that of pleurodynia, is increased by pressure, movement, inspiration, or coughing, but which differs in being associated with more or less pyrexia, and if the inflammation be at the base of the right pleura, the pain may be indistinguishable from that of peri-hepatitis. It is probable indeed that in some of the cases of so-called diaphragmatic pleurisy the inflammation is on the under surface of the diaphragm rather than on the upper, while observations in the dead house leave little doubt that in many there is inflammation on both sides. Dulness on percussion or pleuritic friction over the base of the lung, or the concurrence of pneumonia, will often assist the diagnosis in favour of pleurisy; but in slight cases of diaphragmatic pleurisy there may be neither dulness nor friction, and peri-hepatitis will occasionally give rise to a rubbing sound during the respiratory movements. (See Case XVII., p. 90.)

4. *Gastric Dyspepsia.*—Patients very commonly refer the pain arising from various disorders of the stomach to disease of the liver. The liver is said to be out of order, when the stomach or the duodenum is the organ really at fault. On the other hand, we have found that attacks of pain are often put down to gastralgia, which are in reality slight attacks of biliary colic (p. 339).

a. Pain coming on after food may be due to derangement of the stomach or duodenum, or more rarely to congestion of the liver; but the liver is not likely to be the source of the pain, unless there be tenderness on pressure in the right hypochondrium and those other signs of congestion of the liver which I have already described to you (p. 132).

b. Attacks of severe pain (gastrodynia) occur in the stomach independently of food, and may simulate hepatic colic or hepatic neuralgia. From hepatic colic it will be distinguished by its situation, by its being often accompanied by the eructation of watery fluid (pyrosis), and by the absence of jaundice, or of bile-pigment in the urine, or of tenderness on pressure over the gall-bladder (see p. 339). Neuralgia of the stomach can only differ from hepatic neuralgia in the situation of the pain.

5. *Intestinal Colic* resembles hepatic colic in there being paroxysms of severe abdominal pain with vomiting and shivering, but without acute tenderness on pressure. It differs from it in—

a. The situation of the pain, which is referred to the umbilicus rather than to the epigastrium and right scapula.

b. The absence of jaundice, or of bile-pigment in the urine.

c. The absence of any tenderness on pressure over the fundus of the gall-bladder.

d. The circumstances under which it occurs, viz.: constipation; some obvious error in diet; the presence of lead in the system, indicated by the blue line along the margin of the gums, by a history of lead colic or palsy, or by the patient's occupation. Both may occur in persons of a gouty habit.

6. In *Renal Colic* there is also severe paroxysmal abdominal pain with vomiting and shivering, but—

a. The pain is referred chiefly to one kidney, and thence it shoots down the corresponding thigh and down to the corresponding testicle, which is retracted.

b. There is no jaundice.

c. There is tenderness over the kidney, but not over the fundus of the gall-bladder.

d. The urine contains blood and crystals, which may require the microscope for their detection, or there is a previous history of hæmaturia, or of the passage of a calculus by the urethra.

Keeping in mind these sources of fallacy, we may proceed to consider the varieties and causes of pain which is justly referable to the liver. It will suffice in most instances if I merely mention the diseases in which the pain occurs, inasmuch as I have already described their leading characters to you in former lectures. I may remind you, however, that certain diseases of the liver are characterised by a remarkable immunity from pain, and more especially the waxy liver (p. 33), the fatty liver (p. 48), simple hypertrophy (p. 54) and atrophy (p. 257), and hydatid tumour (p. 58).

Pain having its source in the liver may be said to present three varieties.

I. First, there is a pain which is very severe, comes on in paroxysms with distinct intermissions, and is associated with little or no tenderness, except in the region of the gall-bladder, and with little or no fever, but is often attended or followed by jaundice. Pain answering to this description results from:

1. The presence of gall-stones or other foreign bodies in the bile-ducts (see pp. 337, 344, and 345).

2. Obstruction of the common bile-duct by a duodenal ulcer (see p. 349).

3. An aneurism of the hepatic artery (see p. 356).

4. Hepatic neuralgia. This has been described by Andral, Frerichs,[1] Budd,[2] Anstie,[3] and other writers, and although it is probable that in most of the reported examples, and especially in those where there has been jaundice, the pain has been due to gall-stones which have not been passed (see p. 339), or which have escaped observation, there are others which, occurring in nervous persons or hysterical females, often at tolerably regular intervals of about a month, associated with other nervous symptoms and with no jaundice, seem really to be instances of neuralgia of the hepatic plexus of nerves. I must add, however, that I do not remember to have met myself with an unmistakable example of hepatic neuralgia, and as an illustration of the risk of fallacy in its diagnosis I may refer to a case which I believed to be of this nature, and on which I gave a clinical lecture some years ago.[4] The patient was liable to severe paroxysms of pain in the right hypochondrium and shooting back to the scapula, coming on about once a month suddenly and lasting an entire day, occasionally accompanied by vomiting, but never followed by jaundice or any tenderness over the gall-

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[1] Frerichs, Dis. of Liver, Syd. Soc. Ed., ii. 548.

[2] Budd, Dis. of Liver, 3rd ed. p. 380.

[3] On Neuralgia, London, 1871, p. 62.

[4] The case was recorded as one of 'Hepatic Neuralgia' in the first edition of these Lectures, Case XC., p. 497.

bladder. These paroxysms continued to recur for several years, until at length their real nature was revealed by their being accompanied by hæmaturia and the passage of oxalates in the urine. Notwithstanding the unusual situation and radiation of the pain, the paroxysms were probably nephritic.

II. Secondly, there is a pain in the liver which is not severe and is often described rather as a feeling of weight or distension, which is often associated with pain in the right shoulder, which does not intermit, and which is slightly increased by pressure, or by lying on the left side,[1] or after meals. This pain is often associated with slight febrile disturbance and more or less jaundice. Pain of this description is observed in:—

1. The various forms of congestion of the liver (p. 132).
2. The early stages of hepatitis (pp. 139, 182).
3. Catarrh of the bile-ducts (p. 152).
4. Obstruction of the common duct followed by great accumulation of bile within the liver (p. 161).
5. The pain in acute atrophy (p. 261) partakes somewhat of this character.

III. A third form of pain is constant and severe, is greatly aggravated by pressure, movement, or coughing, and is associated with more or less fever, but not often with jaundice. This pain may be attended by a grating sound like pleuritic friction and by dry cough, but the case is distinguished by the marked tenderness over the liver generally, and by the pain being greatly increased when the patient lies on the left side as well as on the right. This is the pain of peri-hepatitis, which, as I have already told you, although sometimes a primary affection, and then usually syphilitic, is more commonly secondary to other diseases of the liver. Peri-hepatitis accounts for the acute pain that occurs in diseases such as cirrhosis (p. 281), waxy enlargement (p. 33), or hydatid of the liver (p. 58). whose natural course is painless; or it may aggravate the already existing pain of abscess (pp. 165, 183), or cancer (p. 210). Whatever may be the primary disease of the liver, pain of the character last described always indicates inflammation of the capsule, and its supervention sometimes furnishes indications of some importance. Its occurrence, for instance, in a case of hydatid of the liver would indicate that the hydatid was about to burst or contract adhesions to some of the adjacent viscera.

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[1] Great enlargement of the liver from any cause will also give rise to a dragging pain, when the patient lies on the left side.

In former lectures I have brought under your notice many examples of diseases of the liver in which pain was a prominent symptom, and to which I beg again to call your attention.

B. PATHOLOGICAL CONSEQUENCES OF GALL-STONES.

In what remains of this lecture I wish to say a few words on the subject of gall-stones and diseases of the gall-bladder. In a former lecture, when speaking of the causes of jaundice, I described to you at some length the consequences of gall-stones passing along the bile-ducts, or becoming impacted in the common duct, but in doing so I by no means exhausted their clinical history. I purpose now supplying this deficiency by calling your attention to the different situations in which gall-stones are found, to the erratic courses which they sometimes take in their endeavour, so to speak, to escape from the body, and to the varying symptoms and dangers which may result from them accordingly. Those of you who wish to have further information on the subject of gall-stones I would refer to the elaborate Memoir of M. Fauconneau-Dufresne, which obtained the prize of 1,500 francs from the French Academy of Medicine in 1847.[1]

1. *Gall-stones may be retained in the Gall-bladder.*—The gall-bladder is the portion of the biliary passages in which calculi are found most frequently and in largest quantity, and there is abundant evidence that they may exist there for a long time without giving rise to symptoms of any sort. You will constantly find concretions in the gall-bladder after death in the bodies of persons who during life have exhibited no symptoms of their presence. But occasionally, when the concretions are numerous and large, they cause a sensation of uneasiness, weight, tension, or dragging, in the region of the gall-bladder, which is usually worse after meals, after any violent muscular exertion, or after driving over rough roads. I have now under my care a lady who has suffered from gall-stones, and who frequently complains of a sensation of a heavy weight rolling from side to side in the situation of the gall-bladder when she turns in bed. Fauconneau-Dufresne quotes a precisely similar

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[1] La Bile et ses Maladies, Mém de l'Acad. Roy. de Méd. 1847, xiii. p. 36.

case from Fabricius Hildanus.[1] Gall-stones in the gall-bladder also now and then cause vomiting and other derangements of the stomach, and their pressure on the stomach has been known to determine all the symptoms of stricture of the pylorus. It is very possible also that in persons of a nervous constitution they may be a centre of irritation, from which may arise uneasy sensations and symptoms of actual disease in distant parts of the body, with great mental depression and hypochondriasis. Several cases which have been under my care have served to impress me strongly with this opinion.

When the gall-bladder is full of concretions, it sometimes forms a tumour which is appreciable through the abdominal parietes, and the real nature of which may be recognised by its hard and resisting character. On palpation, also, a peculiar crackling sensation, or an actual sound, is in rare instances elicited, which has been aptly compared to that produced by grasping a bag of hazel-nuts or by rolling about small pebbles in the mouth. The stethoscope in these cases may afford material assistance in the diagnosis.

Gall-stones retained in the gall-bladder may also excite inflammation and ulceration of the mucous membrane, and ulterior consequences to be presently referred to.

2. *Gall-stones may become impacted in the neck, or in the cystic duct, of the gall-bladder.*—When a concretion passes from the gall-bladder into the cystic duct, it usually gives rise to vomiting and the symptoms of biliary colic already described (p. 337), but so long as it does not advance beyond the cystic duct there is no jaundice. Sometimes the calculus never reaches the common duct; it becomes impacted in the cystic duct, or it drops back into the gall-bladder, and in either case the colic may cease without there having been any jaundice. At the same time in post-mortem examinations one occasionally finds the neck of the gall-bladder obstructed by an impacted gall-stone, although no symptom leading to the suspicion of gall-stones has ever been observed during life. Permanent obstruction of the cystic duct by a calculus may lead to inflammatory enlargement of the gall-bladder, to the consideration of which we shall presently return.

3. *Gall-stones may form in the radicles of the hepatic duct in the interior of the liver.*—It is not often that concretions form in the bile-passages within the liver, inasmuch as the bile is not

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[1] Fauconneau Dufresne, op. cit. p. 274.

here subjected to those conditions of concentration and repose, which contribute so much to their formation in the gall-bladder. They have sometimes been found, however, in the dilated bile-ducts within the liver in cases of obstruction of the ductus communis choledochus, and there are also a few cases on record where biliary concretions have been found within the liver independently of any such obstruction. These concretions may be numerous, but very small, constituting what has been called 'biliary gravel;' at other times they are large and branched like a piece of coral, as in this drawing which is copied from one of Cruveilhier's Pathological Plates.[1] Chopart met with a case where the liver contained so many concretions that it could not be cut with a scalpel.[2] These concretions in the liver may induce partial obstructions of bile and dilatation of the ducts; sometimes they become enclosed in firm fibrous cysts, shut off from the bile-ducts; and at other times they cause cystic dilatation and ulceration of the ducts and multiple abscesses in the liver. A case has been recorded by Dr. Tuckwell where a large abscess in the right lobe of the liver formed in this way perforated the diaphragm and caused empyema and gangrene of the right lung.[3]

The symptoms of intra-hepatic concretions are usually obscure. They do not cause jaundice nor enlargement of the liver, and the smaller concretions may cause no pain. But now and then they give rise to a feeling of weight, or of dull pain, in the region of the liver, with sudden attacks of sharp cutting pain or violent colic shooting from the right hypochondrium through the chest or to the hypogastrium, while in other cases they have occasioned attacks of rigors[4] followed by heat and sweating, which have simulated ague. Fauconneau-Dufresne records at length a case which was diagnosed by Professor Trousseau, from his finding biliary concretions in the motions associated with attacks of pain like that which I have described.[5]

4. *Gall-stones may be impacted in the hepatic duct, before its junction with the cystic duct*, but this is a rare occurrence. A concretion in this situation must be derived from the ducts within the liver, and if a calculus has succeeded in escaping from the small ducts in the liver, it is not likely to encounter any serious obstacle to its progress or to cause symptoms of

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[1] Livraison xii. Pl. V.

[2] Fauconneau-Dufresne, op. cit. p. 249.

[3] Path. Trans. 1870, vol. xxi. p. 223.

[4] Frerichs, op. cit. ii. 516.

[5] Op. cit. p. 270.

any importance in its passage through the larger hepatic duct. It is not surprising then that there are very few cases on record where a large gall-stone has been found obstructing the hepatic duct after death; but where this does happen during life, there would be jaundice, enlargement of the liver, vomiting, biliary colic, and those other symptoms of obstruction of the common duct which have been already detailed to you (p. 337). There would, however, be no enlargement of the gall-bladder, as when the common duct is obstructed.

5. *Gall-stones may be lodged in the ductus communis choledochus.*—This, in fact, is one of their most common situations, and they find their way here from one of two sources, either from the intra-hepatic ducts, or more commonly from the gall-bladder. As a rule they are discharged sooner or later into the duodenum, their passage being marked by those symptoms of biliary colic which I have already endeavoured to describe to you. The passage of the concretion through the cystic duct gives rise to severe colic, but when it enters the common duct jaundice is added to the former symptoms, and, in consequence of the larger size of the common duct, the pain usually abates in severity, again to return with even increased intensity on the arrival of the concretion at the narrow duodenal orifice, on dropping through which it sometimes ceases suddenly, as if by enchantment. More rarely the calculus is firmly impacted in the common duct and becomes one of the causes of permanent jaundice. A rough angular stone will experience more difficulty and cause more pain in passing along the duct, than one which may be larger but is rounded and smooth, but at the same time it is more likely to permit a little bile to trickle past it. Now and then the common bile-duct is found dilated into a large pouch containing numerous calculi, but at the same time allowing the bile to pass on to the bowel. I recently met with this condition in the body of a lady whose case I shall take another opportunity of relating to you (Case CLXV), and a similar appearance is figured in one of Cruveilhier's Plates.[1] Morgagni long ago reported a case where the common bile-duct was dilated to the size of a small bottle and was full of calculi,[2] and more recently a case has been recorded by

[1] Anat. Path. Livraison xxix. Pl. IV. fig. 3.

[2] Trousseau, Clin. Méd. ii. 532. The common bile-duct in this case is commonly quoted as having been dilated to the size of the human stomach, owing, as Dr. Wickham Legg has pointed out, to Morgagni, in quoting the case from Schenk, having converted the words 'instar utriculi' into 'instar ventriculi.'

Frerichs in which it was converted into a sac measuring 8 in. in length and 5 in width.[1]

6. *Gall-stones may excite inflammation and ulceration of the lining membrane of the gall-bladder or bile-ducts, and may thus lead to perforation and peritonitis or pyæmia.*—While still in the gall-bladder or in any part of the biliary passages, gall-stones may be followed by other consequences besides those already mentioned. Their pressure or irritation may excite inflammation of the mucous membrane with which they are in contact, and this may spread through the entire biliary passages. You will remember that, when lecturing on inflammation of the biliary passages, I referred to the irritation of gall-stones as one of its causes (p. 154), and that not long ago we had an instance of inflammation from this cause in the wards (Case LXVI. p. 162). Occasionally, and more especially when the cystic duct is obstructed, inflammatory products accumulate in large quantity in the gall-bladder from which all the colouring matter of the bile is absorbed, and the gall-bladder becomes converted into a large, painful, fluctuating tumour, simulating an abscess, which may burst in different directions, or may be opened by the surgeon, as in Case CLXX. The pressure of gall-stones may also cause ulceration, which may not be limited to the mucous membrane, but may perforate the entire coats of the gall-bladder or of a bile-duct, and induce fatal peritonitis from the escape of bile or of the concretions themselves into the peritoneum. I show you here some gall-stones removed from the body of a lady lately under my care, who died suddenly of peritonitis from ulceration and perforation of the gall-bladder (see Case CLXIV.). A similar case has been reported by Dr. J. W. Ogle,[2] and you will find in Fauconneau-Dufresne's memoir an observation quoted from Dr. Wolf, where death from peritonitis was due to complete rupture of the hepatic duct.[3] Trousseau also mentions a case where a calculus and a considerable quantity of bile escaped into the peritoneum through a rupture in the ductus communis choledochus.[4] In some of these cases the rupture seems to have been independent of ulceration, and to have been the result merely of undue pressure upon the coats of the biliary passages, which have perhaps been weakened by inflammation or fatty degeneration.[5] Fatal peritonitis in these

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[1] Op. cit. ii. 469.

[2] St. Geo. Hosp. Rep. iii. 189.

[3] Op. cit. p. 273.

[4] Clin. Méd. ii. 533.

[5] See Budd, op. cit. p. 234.

cases is sometimes prevented by the formation of adhesions and the establishment of biliary fistulæ, or the calculus may escape from the gall-bladder and become encysted in its vicinity by organised lymph.[1] Cases have also been recorded where gall-stones have escaped by ulceration from the biliary passages into the substance of the liver, and been there found in the interior of abscesses which communicated with the biliary passages.[2] More commonly it happens that the ulceration or sloughing produced by the pressure or irritation of the gall-stones contaminates the portal blood, and leads to the development of multiple pyæmic abscesses in the manner already explained to you. I would recall to your recollection, by way of illustration, the instructive case of Mrs. —— (Case LXXI., p. 173), which was related to you in a former lecture. You will thus see that an attack, which in the first instance may appear to be nothing more than one of colic from gall-stones, may suddenly and unexpectedly assume all the symptoms of peritonitis or pyæmia, and prove fatal; and the practitioner, if he be not on his guard in giving a prognosis, may incur unmerited discredit.

7. *A gall-stone which has reached the bowel may be voided per anum.*—Once arrived in the bowel, the natural exit of a gall-stone from the body is by the anus, and this is the termination which it is the great object of treatment to promote. As a rule all symptoms cease as soon as the concretion leaves the bile-duct, but occasionally, when the concretions are large, or when numerous concretions become cemented together by fæcal matter into one large mass, their discharge per anum is preceded by severe colic, vomiting, and great prostration. Biliary calculi voided per anum are sometimes remarkable for their size. Fauconneau-Dufresne[3] and Frerichs[4] have collected the notes of several cases where they were as large as a pigeon's or even a hen's egg, and in the Museum of the Royal College of Surgeons are two calculi passed per anum, one of which measures 1¾ in. by 1½ in., and the other is nearly 2 in. in length.[5] In 1868 Dr. Hilton Fagge exhibited to the Pathological Society two specimens of oval biliary concretions passed per anum

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[1] For example, see Simon, Path. Trans. v. 157; and Sharman, Med. Times and Gazette, 1859, i. 274.

[2] Fauconneau-Dufresne, op. cit. p. 340; and Tuckwell, Path. Trans. 1870, xxi. 223.

[3] Fauconneau-Dufresne, op. cit. p. 319.

[4] Frerichs, op. cit. ii. 523.

[5] Catalogue of Calculi, pp. 168, 176.

whose long and short diameters measured about 2¼ in. by 1⅕ in.[1] In the same year one of the largest gall-stones on record was figured in the Lancet by Mr. J. Blackburn. It was 3⅜ in. long, and 1½ in. wide, and weighed 1 oz. 6 dr., and was said to have been passed per anum, 'without any aggravation of the usual signs of gall-stones.'[2] In most instances of these large concretions it is probable that they have found their way into the bowel, not through the bile-duct, but through a biliary fistula from the gall-bladder, although Rokitansky observes that owing to the extreme distension which the biliary passages are capable of, calculi of the size of a hen's egg are enabled to pass through them.[3]

8. *Gall-stones may be vomited from the stomach.*—In rare instances gall-stones are expelled from the body by vomiting. Two instances are reported by J. L. Petit, in one of which the calculus was 2½ in. long.[4] Eight others have been collected by Fauconneau-Dufresne;[5] one is reported in the Pathological Transactions by Mr. Jeaffreson of Framlingham, where the concretion was larger than a nutmeg;[6] and one by Dr. E. J. Miles, where sarcinous vomiting ceased on the ejection from the stomach of two large gall-stones.[7] The expulsion of the stones has usually been preceded for several days by severe pain in the stomach, and accompanied by violent and protracted vomiting. It has been the custom to account for these cases on the supposition of an antiperistaltic action of the duodenum, but in most of the recorded instances it is probable that the calculi have found their way into the stomach by a direct fistular communication with the gall-bladder. This view is favoured by no mention being made of jaundice in many of the cases; by several calculi being vomited in some,—in one as many as twenty; and by one patient vomiting a calculus on three different occasions at intervals of several years.[8] The

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[1] Path. Trans. xix. 254.

[2] Lancet, 1868, ii. 784. For mention of a larger stone, see p. 495.

[3] Path. Anat., Syd. Soc. Transl. ii. 165.

[4] Mém. de l'Acad. Roy. de Chir. 1743, vol. i. p. 308.

[5] Op. cit. p. 306.

[6] Vol. xii. p. 129.

[7] Lancet, Jan. 19, 1861.

[8] Since this lecture was delivered, the sequel of Mr. Jeaffreson's case has been published by his brother, Dr. H. Jeaffreson, from which it would appear that the patient died shortly after the report of her case to the Pathological Society, and that the gall-stone was found to have escaped by ulceration into the stomach. Brit. Med. Journ. May 30, 1868.

possibility, indeed, of a large calculus passing backwards through the pylorus is very doubtful.

9. *Gall-stones after entering the bowel may be impacted and become a cause of intestinal obstruction.*—I show you here a specimen, consisting of a portion of the ileum with a large gall-stone tightly fitted into its interior like a cork, which was obtained from the body of a patient who died in this (Middlesex) hospital a few years ago with all the symptoms of intestinal obstruction (Case CLXVI.), and medical literature contains the records of many similar cases.[1] The intestine in these cases is greatly distended above the obstruction, but contracted and empty below. The impaction of the calculus in the small intestine is marked by obstinate constipation, vomiting, first of food, then of bile, and lastly of stercoraceous matter, pain and tenderness of the abdomen and other symptoms of peritonitis, which symptoms continue until death, or until the concretion escapes into the large bowel. Although these cases are often fatal, it is satisfactory to know that in several of the 25 cases of which I have collected the notes, the concretion has been discharged per anum and the patient has recovered, even after stercoraceous vomiting had set in. In one of the cases (Dr. Omond's) stercoraceous vomiting had lasted upwards of three weeks, and yet the patient recovered.

A concretion which has found its way through the bile-duct is not likely to become arrested in the bowel. Accordingly, in most cases of obstruction of the bowel by a biliary calculus this has passed directly from the gall-bladder into the bowel by a fistulous opening, and thus there may be no previous history of jaundice to assist in diagnosing the cause of the obstruction. For example, this was what had occurred in the case of which I have shown you the specimen. On the other hand, you will remember Rokitansky's statement that a calculus as large as a hen's egg may pass through the bile-ducts. Abercrombie also relates the case of a man who after many attacks of colic followed by jaundice, died on the fifth day of a similar attack, with the addition of symptoms of ileus. A biliary calculus, measuring 4 by 3½ in. in circumference, was found plugging the ileum; the gall-bladder is spoken of as inflamed and partially disorganised, but no mention is made of a fistula, while the common duct was patent and easily admitted the finger.[1] In another case, reported in the Pathological Transactions (xv. 106), where the concretion in the ileum had a circumference of four inches, there had been no jaundice, and yet there were no adhesions about the gall-bladder, which, however, was not carefully examined. In Mr. Le Gros Clark's case, the ileum was obstructed by two large gall-stones 1 in. in length and 4 in. in circumference, and although there had been no jaundice it is stated that there was nothing to indicate that the concretion had ulcerated through into the duodenum from the gall-bladder. It is difficult to account for these cases, if they have been correctly observed. We know, however, that biliary concretions may acquire increased dimensions in the bowel from the deposit of fæcal matter, and that sometimes they form the nucleus of large intestinal concretions;[2] and also that enormous biliary calculi may be lodged for a long time in pouches of the duodenum or jejunum, without causing obstruction of the bowels, which, however, might ultimately ensue.[3]

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[1] Five cases collected by Fauconneau-Dufresne, op. cit. p. 311; two observed by Cruveilhier, Traité d'Anat. Path. ii. 543; two by Frerichs, op. cit. ii. 524; one by Oppolzer, Zeitsch. d Gesellschaft d. Aertze in Wien, Nov. 1860; one by Sir Thos. Watson, Lect. Pract. Phys. 3rd ed. ii. 465, which is the same as Mayo's case included in Fauconneau-Dufresne's five; one by Dr. Omond, Lond. and Med. Surg. Journ. 1836; one by Peacock, Trans. Path. Soc. i. 255; one by Pye Smith, ib. v. 163; one by Baly ib. x. 185; one by Potts, ib. xv. 105; one by Murchison, ib. xx. 219; one by Dr. Ezra Palmer, Records Med. Soc. Bost. U.S. vol. iii. 106; one by Dr. P. Campbell, Med. Times and Gaz. 1870, i. 335; one by Mr. Le Gros Clark, Med. Chir. Trans. 1872, lv. 1; one by Dr. J. S. Gray, Clin. Trans. 1873, vi. 193; one by Dr. Crichton Browne, Brit. Med. Journ. 1875, i. 345; one in Coll. Surg. Mus. Path. Ser. No. 1182; one in Barth. Hosp. Mus. Ser. xvi. No. 84. See also Cases CLXVI. and CLXVII. and the references to Cystico-duodenal fistulæ, p. 497.

Ileus from gall-stones is distinguished from other forms of intestinal obstruction by its usually occurring in females of advanced age, by a previous history of hypochondriac inflammation, by the intensity of the pain, the incessant and severe vomiting, the frequent and intermittent attacks which sometimes seem to indicate the obstruction of the stone here and there in its passage down the intestine, and the rapidity with which the last attack often ends in death.[4]

Several cases have been recorded where the rectum has been obstructed, just above the sphincter ani, by a large biliary concretion, or by several concretions cemented together by fæcal matter. Obstinate constipation and considerable pain have been the result, but under suitable treatment the concretion has generally been voided per anum and the patient has recovered.

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[1] Diseases of Stomach, 3rd ed. p. 127.

[2] See Dr. P. H. Watson, Ed. Med. Journ. May 1868, p. 989.

[3] See Duffin, Lancet, May 27, 1848, and G. Harley, Path. Trans. viii. 235.

[4] Brinton on Intestinal Obst. 1867, p. 75.

10. *Gall-stones may excite ulceration or gangrene of the bowel, and may even escape from it by a perforation into the peritoneum or externally.*—In obstruction of the small intestine by biliary concretions, death usually occurs before there is time for perforation. Perforation is most apt to happen when the concretion is situated in a portion of the bowel where it can remain for a long time without causing obstruction. Thus several instances have been noted where biliary concretions in the cæcum have caused ulceration, gangrene, perforation, and fatal peritonitis. In rare cases gall-stones have been known to enter the appendix vermiformis, and like other foreign bodies, or concretions formed in the part, to cause ulceration, perforation, and fatal peritonitis. In most of the cases where biliary concretions either in the cæcum or in the appendix have been recorded as producing perforation, it may be doubted if the concretions were not intestinal rather than biliary.[1] Budd, however, observed a case where a gall-stone in the vermiform appendix caused perforation and fatal peritonitis,[2] and another patient died from the same cause under the care of Trousseau.[3] The specimen of a similar case is preserved in the Museum of St. Bartholomew's Hospital; [4] and some years ago a case was related by Dr. Adolphe Sirey where a circumscribed abscess formed round the appendix, and where the concretion ultimately found its way out by an ulcerated opening through the abdominal wall.[5] Lastly, Dr. Horace Jeaffreson has recorded a case where two large gall-stones became shelved in a slightly pouched portion of the ileum just above the valve, and eventually set up irritation and ulceration, which resulted in perforation and fatal peritonitis, and where one of the gall-stones was found to have escaped into the peritoneum.[6]

11. *Gall-stones may lead to fistulous communications between the biliary passages and adjacent parts within the abdomen.*—Adhesions form between the gall-bladder and some adjacent

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[1] This remark, I suspect, applies to most, if not all, of the cases collected by Fauconneau-Dufresne. Op. cit. pp. 313, 316.

[2] Dis. of Liver, 3rd ed. p. 378.

[3] Clin. Méd. ii. 536.

[4] Ser. xvi. No. 65.

[5] Med. Times and Gaz. 1859, ii. 372.

[6] Brit. Med. Journ. May 30, 1868, p. 531.

viscus, and a communication is then established by ulceration advancing from the gall-bladder, or by gangrene.

a. Fistulæ into the Stomach resulting from gall-stones have been already referred to. Cruveilhier observed a case where a fistula between the gall-bladder and the stomach was found closed by a gall-stone.[1] Oppolzer met with a case where the opening was found close to the pylorus,[2] and two others are referred to by Frerichs.[3] You have lately seen a case in which there was a fistulous opening between a dilated bile-duct in the liver and the stomach (Case CXIX., p. 372). I have also told you, that it is probably in consequence of this lesion that gall-stones are occasionally expelled by vomiting. Cruveilhier, indeed, is of opinion, that the very circumstance of a gall-stone being vomited is a positive proof of the existence of such a fistulous communication.

b. Fistulæ into the Duodenum are not very uncommon and are almost invariably due to ulceration or gangrene of the biliary passages excited by gall-stones. The specimen which I hold in my hand is a good example of this fistula and was obtained from the case I have already referred to, where death was due to ileus from the impaction of a large stone in the small intestine (Case CLXVI.). I have collected from different sources the notes of 34 cases in which a similar fistula existed,[4] and in the majority of them the cause of death was the obstruction of the small intestine by a large biliary calculus which had escaped by the abnormal passage from the gall-bladder. In several, however, a large calculus was passed per anum after symptoms of obstruction (Case CLXVII.), and, if the patient survive long the formation of the fistula, it is possible that this may be overlooked after death, and that thus the lesion may be more common than is usually supposed (see Case CLXVIII.). The opening in the biliary passages is almost always at the fundus of the gall-bladder, but occasionally, as in a case reported by Frerichs, it is in the common bile-duct;[1] that in the duodenum is in its third or lowest division. The size of the opening varies according to that of the stone which has been transmitted, and to the period which has elapsed between that occurrence and death. The symptoms attending the formation of these fistulæ are sometimes obscure. Those which have been noted consist mainly in vomiting, with signs of local peritonitis in the region of the gall-bladder. Frerichs in one of his cases observed hæmatemesis and bloody stools. Jaundice is rarely present, for though the cystic duct may be obstructed, the common duct is usually free. It is only occasionally that the diagnosis will be assisted by a previous history of gall-stone colic with jaundice. After the formation of the fistula, there may be nothing to indicate its existence. Unless the gall-stone be large enough to cause obstruction of the bowels, the fistula may cause no symptoms of importance and a biliary calculus may be passed from the bowel with little trouble, which by the route of the bile-duct would have caused tremendous suffering; and thus we have an explanation of a statement of certain early writers, to the effect that small gall-stones cause more pain in their escape from the body than large ones. But if there be permanent obstruction of the common duct and the cystic duct be free, the jaundice which has existed prior to the fistula will disappear, and the motions, unless there be obstruction of the bowels, will contain bile. Thus in Trousseau's case bile escaped through the fistula into the bowel after the common bile-duct had become obliterated. In many cases no doubt the fistula closes, as in Case LXXI. (p. 174).

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[1] Traité d'Anat. Path. ii. 541.

[2] Zeits. der Gesellsch. d. Aerzte in Wien, Nov. 1860.

[3] Op. cit. ii. 525.

[4] Eight cases collected by Fauconneau-Dufresne, op. cit. p. 336; two observed by Cruveilhier, Traité d'Anat. Path. ii. 543; two by Oppolzer, Zeitschr. der Gesellsch. der Aerzte in Wien, Nov. 1860, p. 767; two by Frerichs, op. cit. vol. ii. Obs. 74 and 75; one by Duffin, Lancet, May 27, 1848; one by Blagden, referred to by Duffin, but reference given is wrong; one by Peacock, Trans. Path. Soc. i. 255; one by J. W. Ogle, ib. v. 161; one by Pye Smith, ib. v 163; one by Baly? ib. x. 185; one by Dr. Ezra Palmer, Records Med. Soc. Boston, U.S. vol. iii. p. 106; one by Dr. Crichton Brown, Brit. Med. Journ. 1875, i. 345; one by Trousseau, Clin. Lect., Syd. Soc. Ed., iv. 251; three by Dr. J. W. Ogle, St. George's Hosp. Rep. vol. iii.; my own Case CLXVI.; one in Mus. Col. Surg. Path. Ser. No. 1460; two? in Barth. Hosp. Mus. Ser. xvi. No. 84 and ser. xix. No. 11; one in St. Thos. Hosp. Mus. No. 1412, and referred to in South's edition of Chelius' Surgery, i. 716; one in Mus. King's College, Lond. Dig. Syst. Nos. 57, 259, and 272; one in Charing Cross Hospital Mus. G. 3; one in Mus. of Med. Soc. of Boston, U.S. No. 565, New Eng. Journ. of Med. and Surg. 1825.

c. Fistulæ into the Colon from gall-stones are comparatively rare. Frerichs, Oppolzer, and all writers of authority agree in this statement. The exemption of the colon, as compared with the duodenum in this respect, is probably due to the former bowel being more movable. I have been able to find the records of only nine cases of fistulæ between the gall-bladder and colon. In six of the nine there was cancer of the gall-bladder,[1] one of the six cases occurred in my own practice (Case CLXXVI.). Of the three remaining cases, one, in which the fistula probably resulted from the passage of a gall-stone, is recorded at page 511 (Case CLXVIII.). A second is reported by Dr. J. W. Ogle.[2] The specimen of the third case is preserved in the Museum of St. Bartholomew's Hospital;[3] here there were two fistulæ, one into the small intestine, the other into the colon: a large calculus had passed through the former into the ileum, and another, also large, into the colon and was found in the cæcum. In the body of a patient who died in this (Middlesex) hospital some years ago, I found what was probably a similar condition in a more advanced stage; the gall-bladder communicated with the duodenum, the colon, and the external surface; there was no cancer, but the origin of the disease was probably a gall-stone (Case CLXXI.).[4] But even when the fistula is cancerous it is probable that the ulcerative process leading to its formation is determined by gall-stones. In my own case there had been a previous history of gall-stone colic and jaundice, and in four of the remaining five cases gall-stones were found. In a specimen in the Museum at Boston, U.S., the patient, after symptoms of obstructed bowels, voided per anum, three months before death, a biliary calculus measuring 3¾ in. by 3 in. in circumference, and the signs of cancer did not show themselves till two months later. Of 11 cases of cancer of the gall-bladder collected by Frerichs, gall-stones were present in 9. The symptoms of cystico-colic fistula will be mainly those of the cancerous disease with which it seems to be usually associated. A simple cystico-colic fistula might be expected to cause less disturbance than a cystico-duodenal, as there would be less risk of a large calculus becoming impacted in the large bowel than in the small. Its existence might be so little suspected during life, that possibly it has been sometimes overlooked after death, and its occurrence may be more common than is supposed. (See Case CLXIX.)

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[1] Op. cit. ii. 540.

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[1] Two cases by Fauconneau-Dufresne, op. cit. p. 338; one by Durand-Fardel, Frerichs, op. cit. p. 480; one by Cruveilhier, Traité d'Anat. Path. ii. 543; one by Murchison (see Case CLXXVI., p. 528); one specimen in Mus. of Med. Soc. of Boston, U.S. No. 565. I have found only one instance of a cancerous fistula between the gall-bladder and duodenum, and there also the opening was closed by a calculus. Cruveilhier, op. cit. ii. 543.

[2] St. George's Hosp. Rep. vol. iii. p. 178.

[3] Ser. xvi. No. 84.

[4] Dr. Bristowe has recorded a somewhat similar case where the common bile-duct, in consequence of obstruction from gall-stones, communicated with the duodenum, the colon, and the portal vein. (Path. Trans. vol, ix. p. 285.)

d. Fistulæ into the Urinary Passages.—There are at least two well-authenticated cases on record where biliary calculi have been voided with the urine during life, apparently owing to the formation of a fistula between the gall-bladder and the pelvis of the right kidney.[1] One of the patients passed nine small and four large calculi; the other voided 200 small calculi within a week: in both patients an operation was necessary to remove one of the calculi from the urethra. In both cases the calculi were analysed and found to consist of cholesterin and bile-pigment; and in one the analysis was made by Gmelin, who also found bile-pigment in the urine. Neither of the patients had ever had jaundice; in both the symptoms were those of urinary rather than of hepatic disease; both recovered. In connection with this subject I may call your attention to the case of a patient who recently died of calculous pyelitis while under my care in the hospital, and whose urine always contained a large quantity of cholesterin and pus, although no communication existed between the urinary and biliary passages.[2]

e. Fistulæ into the Vagina.—The only instance with which I am acquainted where a fistula formed between the biliary passages and the vagina is one quoted from Frank by Fauconneau-Dufresne, where an enlarged and inflamed gall-bladder contracted adhesions to a pregnant uterus and burst into the vagina during parturition.[3]

f. Fistulæ into the Portal Vein.—It is a tradition that Realdus Columbus found three gall-stones in the portal vein of Ignatius Loyola, the founder of the order of the Jesuits, which had escaped from the gall-bladder by ulceration:[4] but although it may be doubted whether the concretions in that instance were not phlebolites,[5] there are several well-authenticated examples of fistulous communications between the biliary passages and the portal vein, with the presence in the latter of biliary concretions. Two such cases are referred to by Fauconneau-Dufresne[6] and Frerichs; a third is recorded by Dr. Bristowe in the Pathological Transactions (vol. ix. p. 285), where the common bile-duct also opened into a cavity which communicated both with the duodenum and the colon; a fourth case has come under my own observation (Case CLXXIV.). In these cases the common bile-duct is usually obstructed by a concretion, and the symptoms are those of portal obstruction—ascites or enlargement of the spleen, or both,—or of pyæmia, supervening upon persistent jaundice.

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[1] Fauconneau-Dufresne, op. cit. p. 341, and Gaz. Méd. de Paris, Av. 18, 1840.

[2] This case is recorded in the Pathological Transactions, vol. xix. p. 278.

[3] Fauconneau-Dufresne, op. cit. p. 159.

[4] Frerichs, op. cit. ii. 526.

[5] Thudichum on Gall-stones, 1863, pp. 11, 268,

[6] Op. cit. p. 340.

g. Fistulæ into the Pleura.—One instance is on record of a fistulous communication between the biliary passages and the pleura. This lesion was discovered by Dr. Cayley in the body of a patient who died in the Middlesex Hospital on March 2, 1866, while under the care of Dr. Thompson, and whose case is reported in the Pathological Transactions (vol. xvii. p. 161). All of the bile-ducts were enormously dilated, apparently in consequence of a gall-stone which had been impacted in the common duct, but which had escaped into the bowel before death. The common duct would allow the fore-finger to be introduced into it from the duodenum. The left pleural cavity contained more than a pint of bile mixed with pus, and in the left half of the diaphragm there was a perforation large enough to admit a No. 4 catheter which led into an irregular cavity between the left lobe of the liver and the under surface of the diaphragm, which in its turn communicated with a cystic dilatation of one of the bile-ducts in the liver. The case was also remarkable from the fact that there was no evidence of obstruction of the bile-duct until fifteen days before death, and that there were both the symptoms and the post-mortem appearances of acute or yellow atrophy of the liver.

12. *Gall-stones may be discharged from the biliary passages through fistulous openings in the abdominal parietes.*—The ten gall-stones which are in this bottle were discharged through an opening in the abdominal wall by a lady who has been for several years under my care (Case CLXX.); and I find that in medical literature and in pathological museums there are the records or the relics of at least 86 similar cases,[1] which, with

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[1] Six cases observed or collected by J. L. Petit, Mém. de l'Acad. Roy. de Chir. 1743, ome i. p. 255; one by Haller, Physiologia, Berne, 1764, tome vi. p. 605; eleven cases by Soemmering, De concrementis biliariis corporis humani, 1795, p. 20: one by Saunders, Trans. of Coll. of Phys.; seventeen cases by Fauconneau-Dufresne, exclusive of three cases already reported by Petit, Mém. de l'Acad de Méd. 1847, xiii. 320 and 167; three cases by Oppolzer, Zeits. der Gesellsch. der Aerzte in Wien, Nov. 1860, p. 747; three cases by Walter, Frerichs on Dis. of Liver, Eng. ed. ii. 525; three cases by Budd, Dis. of Liver, 3rd ed. p. 373; three cases in Gazette des Hôpitaux, 1846, Oct. 8, and 1847, p. 212; two by Cruveilhier, Traité d'Anat. Path. ii. 567, 570; two by

few exceptions, have occurred in females of middle or advanced age. No fewer than five have come under my own observation. These fistulous openings are formed in two ways. In some cases the ulcerative process which commences in the gall-bladder or in a dilated duct gradually eats its way through the adherent abdominal wall until it reaches the surface, while in others the gall-bladder or one of the bile-ducts becomes in the first place enormously enlarged from the accumulation of inflammatory products and opens externally, or, being often mistaken for an abscess of the liver, is opened by the surgeon. The external opening is sometimes over the fundus of the gall-bladder, but very frequently it is at the umbilicus, to which it may be directed by the suspensory ligament of the liver; occasionally it is to the left of the middle line, or in the inguinal region, or over the pubes as in a case observed some years ago at Paris in which two biliary calculi were removed from above the clitoris, where they had been encysted in the subcutaneous tissue.[1] In rare cases there are two or more openings. The number of gall-stones discharged by the opening varies from one to upwards of 600. When there is only one, it may be as large as a hen's egg. Fauconneau-Dufresne refers to one which measured 3·15 in. in length by 1·1 in. in width. Calculi may be discharged almost as soon as the opening is formed, or not for years afterwards. The fistula has been known to keep discharging for many months after all the calculi have come away, but usually it soon closes after this has occurred, if there be no bile in the discharge. The fluid which

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Trousseau, Clin. Méd. ii. 534; two by Duplay and Frétin in Archiv. Gén. de Méd. 2me sér. i. 381, and 5me sér. iv. 86; one by Obre, Path. Trans. of Lond. i. 272: one by Simon, ib. v. 156; one by R. Robinson, ib. v. 158; one by Everet, ib. xviii. 120; one by Taylor, ib. xviii. 147; one by Duckworth, ib. xxii. 157; one by Heberden, Comment. 4th ed. p. 210; one by Santo-Nobili, Schmidt's Jahrb. lviii. 62, 1848; one by Schroeder, Prag. Vierteljahrs. xliv. Sup. p. 70, 1854; one by Callaway, Lancet, 1827-8, ii. 296; one by H. C. Stewart, ib. 1849, ii. 294; one by Nesfield, ib. 1870, i. 157; one by W. R. Barlow, Med. Chir. Trans. vol. xxvii.; one by G. Robinson, ib. vol. xxxv. 1852, p. 471; one by Mackinder, Brit. Med. Journ. Dec. 26, 1857; one by Hinton, ib. Aug. 4, 1860; one by Dr. G. H. Phillipson, ib. 1870, ii. 382; one by Alexander, ib. 1876, ii. 397; one by Cockle, Med. Times and Gazette, May 10, 1862; one by Dr. H. Baillie, Ind. Ann. of Med. Sc., vol. xii. p. 295; one by Hertz, Berliner Klin. Wochenschrift, Ap. 7, 1873; one by Krumptmann, Lond. Med. Record, Ap. 30, 1873: five by Murchison (see p. 520); three specimens in Mus. Roy. Coll. Surg. Cat. of the Calculi, pp. 172, 176, 178; one in Mus. of Med. Soc. of Boston, U.S. No. 566; one by McPherson, Amer. Journ. of Med. Sc. vol. lxi. p. 409.

[1] Gaz. des Hôp. Oct. 8, 1846.

drains away from the fistula may be pure bile, the daily quantity of which has been found to vary from 8 oz. to 2 pints.[1] Far more commonly it is pure pus, or a glairy mucus with now and then a little blood. In most cases the cystic duct is obstructed and then no bile can escape by the fistula; in a few cases the common duct only is obstructed, and then the jaundice caused by this obstruction disappears in a great measure on the formation of the fistula, although no bile is present in the stools; in rare cases the gall-ducts are patent and bile passes off both by the fistula and the bowels. After the discharge of a gall-stone the external opening rapidly contracts and is drawn inwards, while the fistulous passage connecting it with the gall-bladder may be several inches in length and may also be extremely tortuous and surrounded with callous induration. Not unfrequently gall-stones become lodged in a cul de sac of the fistula, or may completely obstruct it and lead to the accumulation of matter behind and the formation of fresh abscesses. The adhesions between the gall-bladder and the abdominal parietes may be very extensive, but are often very limited. These fistulous openings are chiefly serious from the inconvenience which they occasion. A large proportion of the patients survive their formation for years and enjoy good health, and in many the fistulæ completely heal.[2] This last event may be predicted with most confidence when there is only one large calculus, when the external opening is directly over the gall-bladder, when the discharge from it contains no bile, and when there is no jaundice. There is little hope of the fistula permanently closing while calculi still remain in the gall-bladder, or if the cystic duct be patent while the common duct is closed. In cases where the quantity of bile lost is great, which are happily rare, the patient may become rapidly reduced in flesh and strength and die of marasmus. When the calculi are many in number, and the fistula is long, tortuous, and surrounded by callous tissue, the openings though small, may continue for years, showing every now and then a tendency to cicatrise, but again enlarging on the escape of a fresh calculus, the passage of which often entails much suffering, although in the intervals the patient enjoys good health.

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[1] Fauconneau-Dufresne, op. cit. p. 323; and cases by Haller, Heberden, Saunders, Barlow, Robinson, Hertz, and Krumptmann. For references, see footnote, p. 501. Also Case CLXXII.

[2] A case has been communicated to me by Mr. James Taylor of Chester in which the fistula became permanently closed after ten weeks. The patient, a female aged 53, had suffered from several typical attacks of biliary colic before the formation of the abscess in the gall-bladder, which was opened artificially.

From this lengthened list of the evils of which gall-stones may be the source, you will learn to avoid the common error of regarding them as a harmless, though perhaps painful, malady, and you will see the necessity in all cases of being guarded in your prognosis.

Treatment of the consequences of Gall-stones.

1. When the symptoms indicate the presence of gall-stones in the gall-bladder, the treatment must consist in those constitutional measures which I have already told you are useful for gall-stones in general (p. 362), and in remedies for correcting any symptoms of indigestion. At the same time the patient should be cautioned as to the risk of a gall-stone being projected into the bile-ducts by sudden or severe muscular exertion, or by driving over a rough road, especially after meals.

2. The measures to be adopted when gall-stones are in the ducts have already been described to you in detail (p. 360). When there is reason to believe that the calculus has entered the bowel, it is well to administer some laxative such as castor oil, with the object of expediting its passage to the anus.

3. When symptoms of inflamed gall-bladder—fever with pain, tenderness, and often a distinct tumour—supervene upon those of gall-stones in the ducts, the treatment may be summed up in the three words which I mention in the order of their importance, viz.:—rest, opium, and leeches. Rest is all-important to promote the formation of adhesions and prevent the extension of the inflammation to the general cavity of the peritoneum. Any sudden movement may produce a rupture in the inflamed and softened gall-bladder and fatal peritonitis in consequence. Opium may be given in large and repeated doses, and I have often known the pain materially relieved by the application of a few leeches below the right ribs. The expediency of puncturing the gall-bladder, when it is very large, will be considered presently.

4. When symptoms of gall-stones in the gall-bladder or ducts, or of inflammation of the gall-bladder, are followed by

those of ileus, although death is too often the result, recovery after the expulsion of a large calculus per anum has been sufficiently frequent to justify us in sparing no effort for the attainment of this desirable end. Warm baths and fomentations, opium and belladonna in full and repeated doses, and copious enemata of warm water and oil are the measures most to be relied on. Gentle pressure and manipulation of the abdomen have been thought in some instances to have displaced the stone. Sir Thomas Watson relates how a lady suffering from ileus experienced a sensation as if this had occurred during her examination by three medical men in succession; while they were still consulting she had a liquid motion precisely resembling what she had last vomited, and next day she voided a gall-stone as big as a walnut.[1] Lastly, in all cases where there is reason to believe that intestinal obstruction is due to biliary concretions, it will be well to examine the rectum. Now and then the obstruction is situated immediately above the sphincter and can be removed by the finger or scoop.

5. Internal biliary fistulæ are beyond the reach of medical or surgical art; but those into the intestines, which are most common, are scarcely dangerous except from their sometimes permitting the escape of a calculus large enough to obstruct the bowels.

6. When external biliary fistulæ discharge one large rounded calculus without facets, and the outer opening is over the fundus of the gall-bladder, they usually heal speedily without any interference. But when the gall-bladder contains many small calculi, and still more when the fistula is long, narrow, circuitous, and surrounded by callous tissue, the opening may remain for years or may never close, and every now and then the fistula is apt to become blocked up by a calculus, the passage of which causes much pain and leads to accumulation of matter behind it. Under these circumstances, the question will arise as to the expediency of dilating or slitting up the fistula to facilitate the extraction of the calculi. This has been done in many cases with a successful result;[2] but on the other hand there are several instances on record, where even slight interference, such as the introduction of a dressing forceps, has brought on fatal peritonitis. There is no general

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[1] Lect. on Pract. of Physic, 5th ed. ii. 549.

[2] See, for instance, a case reported by Dr. H. Baillie, in which 15 gall-stones were removed by operation. Indian Annals of Med. Science. xii. 295.

rule applicable to all of these cases, but the question of operating must be decided by the peculiarities of each case. If, on probing, a calculus can be felt near the outer opening and is long delayed there, it ought to be extracted; but if no calculus can be felt, and still more if the fistula take an inward direction towards the peritoneum, the risk of interference ought to counterbalance the inconvenience of the fistula, and it is better to wait. When pure bile drains away in large quantity from the opening and none enters the bowel, there is little chance of the fistula closing, and it is not desirable that it should, unless, as in Case CLXXII. the obstruction of the common duct were simultaneously removed; but if the common bile-duct be patent, and the patient suffer from the exhaustion consequent on the external drain of bile, the question of closing the fistula may fairly be entertained.

In illustration of some of these evil consequences of gall-stones, I may now bring under your notice the following cases:—

Case CLXV.—*Gall-stones in a Sacculus of Common Bile-duct and in Gall-bladder—Ulceration and Perforation of Gall-bladder—Fatal Peritonitis.*

Mrs. C——, aged 55, had consulted me repeatedly during three years for noises in head and other distressing nervous symptoms, which first appeared after a period of great mental anxiety, and which could only be accounted for by a weak state of circulation, with probably a fatty heart. She had in consequence taken but little exercise, and spent a great part of her time in bed. About end of May 1867 I was called to see her, and found that she had decided jaundice of skin and conjunctivæ, and that urine contained a considerable amount of bile-pigment. Liver also was slightly enlarged. For two or three days she had been suffering from paroxysms of severe pain in right hypochondrium with vomiting. A feeling of soreness remained in intervals of paroxysms and there was slight tenderness below right ribs. Pulse, however, only 72, and skin cool. Under use of warm fomentations, repeated doses of morphia and laxatives, the acute symptoms subsided in a few days; by end of a fortnight, jaundice had disappeared and patient was able to go out.

On June 24 she had a return of severe pain in abdomen. When I saw her on 26th she was again jaundiced and her symptoms differed from those in previous attack in that pulse was 96, skin felt slightly hot, and there was rather more tenderness below right ribs, with tendency to hiccough. Still pain was for most part paroxysmal and relief was again obtained from morphia. For two days she seemed to improve, but on night of 28th she became rather suddenly worse and at my visit on following day she had all the symptoms of acute peritonitis: pulse 136, small and feeble; respirations short, quick, and thoracic; constant vomiting and hiccough; abdomen greatly distended and tympanitic, and acute pain and tenderness, chiefly in left side—constant and aggravated by slightest movement. From this time patient continued to sink until death occurred on night of July 12.

At post-mortem examination two openings were found in fundus of gall-bladder, both with ragged edges and one large enough to admit finger. Through these openings bile had escaped in large quantity into peritoneum. Firm adhesions of great omentum to abdominal wall had directed bile entirely to left side of abdomen, where there were signs of recent peritonitis—intense vascular injection and lymph coloured with bile. Mucous surface of gall-bladder surrounding openings was extensively ulcerated, apparently from pressure of a gall-stone, size of a cherry, which was in immediate apposition and had not escaped into peritoneum. Common bile-duct communicated with a pouch as large as a hen's egg, containing bile and upwards of a dozen polyhedral gall-stones, each about size of half a cherry; but no concretion was found obstructing duct between pouch and duodenum. No abscesses in liver. Heart, kidneys, and liver in a state of fatty degeneration.

CASE CLXVI.—*Fistulous Opening between Gall-bladder and Duodenum—Fatal Obstruction of Small Intestine by a large Biliary Calculus.*

A. McD——, aged 46, was admitted into Middlesex Hospital under care of Dr. Stewart, on Jan. 29, 1856. Her general health had been good, but for many years past she had been of a costive habit, bowels being seldom moved without taking aperient pills. She had been subject to bilious attacks with vomiting of green bitter matter, and to loss of appetite with excessive flatulence after meals, but she never had jaundice.

Twelve days before admission she took two 'antibilious pills,' which acted freely on following day. Vomiting took place at same time and continued with little intermission ever afterwards. Motions and vomited matters were of a green colour. Sleep much disturbed. Two days after this she was suddenly seized with a severe, sharp pain in right iliac region, where tenderness on pressure was still acute; this pain continued with remissions until time of admission. She had no motion for ten days from that time, *i.e.* from the 19th to the 29th of January.

On admission, an injection consisting of a pint of gruel and some castor oil was administered. The whole of it passed up, but it caused great pain and was soon discharged with several large hard scybalous masses. The abdomen then became flaccid; it was slightly dull to right of umbilicus, but elsewhere perfectly resonant. Her countenance was somewhat pinched and anxious and cheeks flushed. Tongue dry and covered with a thick yellow fur on dorsum, moist and clean at edges; thirst very urgent. Pulse 80, very small. Slight sonorous rhonchi on right side of chest; breathing healthy on left side. Heart-sounds normal. A warm bath was ordered, to be followed by poppy-head fomentations to abdomen and an opiate pill.

Jan. 30.—Passed a tolerable night, and notwithstanding a constant sensation of nausea has not vomited since admission. Two pints of injection passed up without difficulty or pain, but after being retained half an hour returned without a trace of fæculent matter. Abdomen is now much more tense and tympanitic and is still painful on pressure.

10 P.M.—Countenance less anxious; thirst very great; nausea continues, but she has not vomited until just now, when she suddenly raised herself on elbow and vomited more than half a pint of dark brown and very offensive stercoraceous fluid; retching continued for several minutes until she had brought up about three pints of this fetid fluid. Pulse 78.

Jan. 31.—There was no recurrence of vomiting till 11 A.M., when she vomited about a pint of fluid having same colour, but without fæculent odour of that brought up yesterday. Abdomen became more and more distended; pain increased; no motion passed; there was frequent retching; exhaustion supervened, and she died rather suddenly this evening.

Autopsy.—Intestines much distended and in some places adherent to each other by recent lymph; great omentum puckered up and adherent to intestines. On separating intestines, a solid body about size and shape of a cork was found to block up small intestine, about middle of ileum, which it fitted like a plug. At point of obstruction gut was bent upon itself, adjacent peritoneal surfaces being slightly adherent by recent lymph. Intestine seemed to become suddenly smaller immediately below obstruction, but above this point it was greatly distended and filled with dark, greenish, fæculent matter, in which were found ten small angular, biliary calculi, of about half size of hazel-nuts; dilated portion of intestine was dark, and when laid open mucous membrane was found much congested and in some parts coated with adherent false membrane; a number of small roundish ulcers were scattered over surface. Obstructing body was discovered to be a large biliary calculus. It was perfectly cylindrical, measuring nearly four inches in circumference, and one inch and a quarter in diameter. Its external surface was uniformly nodulated, the extremities being rather smooth. When divided transversely it exhibited a crystalline appearance.

Below obstruction intestine much contracted and pale throughout containing only a little thickish mucus; colon almost empty.

contained some greenish fæculent matter and one small angular biliary calculus. Gall-bladder firmly adherent to duodenum at point where it turns down to become perpendicular, and a well-defined communication existed between these two parts, large enough to admit a finger easily. Gall-bladder contracted and converted into a small fibrous pouch; there could be no doubt that calculi had passed through this perforation, although opening was now much smaller than calculus causing obstruction. Common bile-duct patent but not dilated; cystic duct closed. Liver weighed 58 oz. and appeared tolerably healthy, though rather dark. Heart, lungs, and kidneys not diseased.

In the next case it is tolerably clear that the small bowel had been obstructed for ten days by a large biliary calculus which had escaped by perforation of the gall-bladder into the duodenum, but that the obstruction was removed and the patient lived for upwards of seven years afterwards. This diagnosis formed during life, notwithstanding that the fæces had not been searched for a stone, was verified by post-mortem examination. In reference to this point I may mention the case of a lady who brought to me some years ago a gall-stone measuring nearly 2 by 1½ in., which she had passed per anum after symptoms of obstruction and inflammation of the bowels. But as regards diagnosis, the case before us was of even greater interest, from the rare conjunction of jaundice from obstruction of the common bile-ducts by gall-stones with ascites resulting from interstitial hepatitis, the latter being itself a result of the irritation set up by gall-stones. Had the patient been seen for the first time in an advanced stage of the malady, with no accurate knowledge of his history, the existence of a tumour would have been the most legitimate inference from such a combination of symptoms (see p. 432). The formation of gall-stones within the bile-ducts, after obliteration of the gall-bladder, is also worthy of notice.

CASE CLXVII.—*Escape of Gall-stone by Ulceration from Gall-bladder into Duodenum—Obstruction of Bowel for ten days—Recovery. Death 7½ years after from Obstruction of Bile-ducts and Ascites.*

On Oct. 23, 1874, Mr. C——, aged 66, consulted me at the request of Dr. Leech of Manchester. Seven years before he had an attack of obstruction of bowels, which lasted ten days and for two days was attended by stercoraceous vomiting, but which ultimately gave way with discharge of most offensive fæces. During five years previous to that illness he had several attacks of very severe spasm, commencing at epigastrium and attended by retching. After the attack of obstruction of bowels he recovered and attended to business, but the paroxysms of colic recurred from time to time, and for six months had been more frequent. In June 1874 he became for first time jaundiced and he had attacks of colic once or twice a week, attended by rigors and followed by fever and delirium, the pyrexia (103°) subsiding in perspiration within 24 hours. Each attack was followed by a marked increase of jaundice, which in intervals almost faded away. Of late these attacks had been less frequent. Motions on several occasions had been searched with care, but no gall-stone had ever been found.

At time of his visit to me, Mr. C. was emaciated and had distinct jaundice of a bronzed hue; intense itchiness of skin preventing sleep. Liver not enlarged; a tender spot, but no prominence, corresponding to fundus of gall-bladder. Much bile-pigment in urine. Diagnosis was: a large calculus in common duct, and previous attack of obstructed bowels, probably due to a large stone which had escaped by ulceration into duodenum.

For six weeks after Mr. C. visited me he seemed somewhat better and he continued to attend to business; the itchiness remained very troublesome, but attacks of pain followed by rigors and fever were less frequent and severe. On Dec. 20 Dr. L. for first time found fluid in abdominal cavity, and a week later there was œdema of legs. Ascites and œdema gradually increased and strength diminished until death on Feb. 23, 1875. During last six weeks of life there was no fever and but little pain. A week before death Dr. L. removed 5 pints of fluid to relieve dyspnœa.

Dr. L. has kindly furnished me with an account of appearances found on post-mortem examination. Several quarts of serum in peritoneum. Mesentery and coats of bowels thickened and œdematous. Diaphragm adherent to upper surface of liver in front part. Transverse colon adherent to under surface of liver, but no sign of ulcer or cicatrix on its mucous surface. On inner surface of duodenum, about 2 in. below opening of bile-duct, was a pouch, at bottom of which was a puckered cicatrix; this corresponded to spot where duodenum was adherent to liver. Opening of common bile-duct in duodenum was distinct and of natural size; but outside duodenal wall duct was enormously dilated and contained a light-brown fluid and one or two gall-stones, which escaped when duct was cut across in removing liver. This part of the duct would easily admit a tube half an inch in diameter, but just where it entered coats of bowel it suddenly contracted so as to admit only a probe. No appearance of cicatrix on its inner surface. Gall-bladder reduced to size of a large nut; cystic duct obliterated. Hepatic ducts in interior of liver greatly dilated and contained 15 gall-stones immersed in a light-brown fluid. All these stones had facets; they varied in size from a hazel-nut to a peppercorn; largest was situated just where hepatic duct entered liver; coats of ducts thickened, but free from ulceration. Walls of portal vein adjacent to dilated ducts also thickened and red, but contained no blood-clots. Liver small and indurated; no abscess in any part of it. Heart and lungs healthy.

There could be little doubt that in the following case the fistulous communication between the gall-bladder and the colon had resulted from the passage of a gall-stone. Although the patient died of epithelioma of the uterus, there was no sign of any new growth in the neighbourhood of the fistula.

CASE CLXVIII.—*Fistulous Communication between Gall-bladder and Colon.*

A woman, æt. 60, died in Middlesex Hosp. on Feb. 7, 1870. She had always enjoyed good health till five months before death, when she was seized with severe abdominal pain, coming on in paroxysms, and attended by nausea and retching. She kept her bed for two days, but had no jaundice, and there was no evidence of her having passed any stone. After this she suffered from a pain in the uterine region, and the immediate cause of death was epithelial cancer of the uterus inducing peritonitis.

At the autopsy the gall bladder was found to be shrivelled into little more than a duct; it contained a little mucus not tinged with bile; the channel of the cystic duct was obliterated and the fundus was inseparably adherent to the transverse colon, with which it communicated by a circular orifice, with smooth well-defined edges and about four lines in diameter. There were signs of old inflammation about the fissure of the liver, the capsule of which was thickened, but the outer surface was smooth. The substance of the liver was firm and fibrous, but there were no cancerous masses either here or in the neighbourhood of the gall-bladder or colon.

Notwithstanding its obscurity, the following case illustrates a sequel of biliary colic to which, so far as I know, attention has not previously been drawn. It is all the more deserving of attention as the patient is an eminent member of the medical profession who has watched and recorded his symptoms with great care and minuteness, and as different opinions of his case have been expressed by the distinguished physicians whom he has consulted. Some of these opinions are worth mentioning. One physician with much experience in Indian diseases ascribed the symptoms to chronic inflammation of the cæcum and ascending colon; but this view appeared to be negatived by the absence of inflammatory matters from the stools, by the persistence of the soreness for years after all purging had ceased, and by its not accounting for the origin of the symptoms in an inflammatory attack during the passage of gall-stones. Sir Thomas Watson, who was consulted in 1867, looked upon the soreness as a neuralgic residue of the gall-stone attacks. Now, I have already had occasion to mention to you that neuralgia is an occasional sequel of biliary colic (p. 338); but here the pain, if indeed I may call it so (for the patient said it was not pain), has not been neuralgic in its character; there has been no spinal tenderness; while neuralgia would not account for the diarrhœa and for the relief of the soreness whenever the patient assumes the recumbent posture. Sir William Jenner, while declining to express any positive opinion, thought that the symptoms might be accounted for by the presence of adhesions between the gall-bladder and surrounding parts, and there is much in this view to commend itself. It accounts for the mode of origin of the soreness and for the influence of posture, but it fails to give a solution of the diarrhœa which immediately followed, and was more or less persistent for nearly four years after, the inflammatory attack. The view which appears to me to offer the best explanation of all the circumstances of the case is, that in the inflammatory attack which occurred in May 1866, not only did the gall-bladder become adherent, but a fistulous passage was established between it and the colon, such as was present in Case CLXVIII. The passage of fresh bile into the colon from the gall-bladder would be more likely to occur when the patient was erect, and might account for the feeling of soreness along the ascending colon and also for the diarrhœa. It might be contended in opposition to this view that no large gall-stone was found in the stools after the inflammatory attack in 1866; but such a stone might have been overlooked, and there is no proof of the necessity of a gall-stone being large in order that it should ulcerate its way from the gall-bladder into the bowel. In reference to the view which I have suggested it is also to be noted, that the last attack of biliary colic in June 1866, about the most severe and protracted which the patient had experienced, was not followed by a trace of jaundice. This is just what might have been expected if the stone had found its way into the bowel through the newly formed fistulous passage. Lastly, the rarity of the patient's symptoms as a sequel of biliary colic is in accordance with the pathological fact already referred to (p. 498), that the fistulæ between the gall-bladder and bowel resulting from gall-stones almost invariably take the direction of the duodenum and not of the colon. This very circumstance, however, renders it impossible to make a positive diagnosis.

CASE CLXIX.—*Repeated Attacks of Biliary Colic followed by persistent Pain in right side and Diarrhœa—Fistula between Gall-bladder and Colon?*

Dr. H——, aged 57, consulted me on two occasions during September 1869. Upwards of three years before he had suffered during three months from a succession of attacks of biliary colic. From March 12 to June 15, 1866, he had in all 19 attacks, lasting 134 hours, and 8 or 10 stones were found in stools. The attacks were attended by vomiting; but so far as he could remember, although on this point he was not absolutely certain, decided jaundice first appeared on May 13, after seventeenth attack of colic, and subsided in a few days. Several gall-stones had been found in stools prior to May 13; a large one was found on May 15, and another on May 19, two days after the eighteenth attack of colic. On May 20 he had a severe rigor, which lasted an hour and was followed by fever, considerable tenderness over liver and abdomen generally, vomiting, and diarrhœa. This attack confined him to bed for a week. The last attack of biliary colic occurred on June 14 and 15, and was one of the longest and most severe. There was no jaundice with this attack, but after it was over a small gall-stone was found in the stools. In all, 8 or 10 stones were found. After these attacks of biliary colic he remained very prostrate for several months, and although then he gradually regained strength, he had suffered ever since from weakness, sleeplessness, depression of spirits, gastrodynia and other dyspeptic symptoms, but particularly from diarrhœa, and from 'a feeling of soreness and distress, not pain,' in region of cæcum and ascending colon. This soreness became permanent; it was never absent, except when patient was in bed or in recumbent posture; and it was so wearing and exhausting that he lost all vigour, and for months at a time he was obliged to give up all work. The patient himself had always associated this soreness and the diarrhœa with the attack of diarrhœa followed by fever and abdominal tenderness, which he had experienced on May 20, 1866.

On careful examination of abdomen, I could discover nothing abnormal. I had no opportunity of seeing the stools; they were loose and did not exceed three or four in the day. I saw several of the gall-stones which had been passed in 1866; they were about size of large peas, nodulated on surface but without facets, and composed of pure cholesterin.

The patient continued without any improvement until March 1870, when he commenced the use of opium. He took two or three grains of opium per diem by mouth, and as the dose was gradually increased he substituted morphia, but he never exceeded three grains in the

24 hours. This treatment was at once attended by manifest advantage. The side-ache was mitigated, though not removed; the diarrhœa permanently ceased; he felt fit for anything and was in every way a changed man. In November 1872 he substituted the hypodermic injection of morphia, which he has persisted with ever since, having injected 4 gr. daily during the last two years. This plan has answered even better. Its invigorating effects have been most remarkable; and it has seemed to reach and control the side-ache in a way that morphia by the mouth never did, so that the patient is able (August 1876) to perform a fair amount of active duty. Nevertheless, when not under the influence of morphia, the side-ache appears to have gained in intensity, and were it not for the morphia would be quite unbearable.

The following case was of great interest from several points of view. It was a good illustration of the large size which the gall-bladder sometimes attains from the effects of inflammation, and it showed how readily this condition may be mistaken for abscess of the liver. Before the passage of the renal calculus, the previous history of the patient and the fact that the symptoms were evidently connected with the right kidney suggested the idea that a biliary calculus might have ulcerated its way into the pelvis of the right kidney and obstructed the right ureter, as in the cases I have already brought under your notice (p. 500), but the analysis of the stone left no doubt that the patient was the subject of both renal and biliary calculi. The concurrence of these two maladies was long ago pointed out by Baglivi and Morgagni,[1] and has recently been made the subject of fresh investigations by Dr. Sénac of Vichy, who found that out of 128 patients suffering from hepatic colic, 98 were either simultaneously, or had been previously, the subjects of lithic acid gravel.[2]

Case CLXX.—*Hepatic Colic—Closure of Cystic Duct—Abscess of Gall-bladder—Discharge of Gall-stones through a fistulous Opening in Abdominal Parietes—Passage of Renal Calculus.*

On June 18, 1867, the Countess ——, about 54 years of age, consulted me on account of a fistulous opening in abdominal parietes, and gave following history of her illness, which was subsequently supplemented by Mr. Bickersteth of Liverpool, who had previously attended her. In previous autumn she had suffered from an attack of biliary colic with jaundice and vomiting, which had passed off after two or three weeks. Towards end of year she had been attacked with persistent [illegible] in region of liver, which gradually increased and was attended by considerable fever and other symptoms of constitutional disturbance, but not by jaundice. Very soon an enlargement was noticed in right hypochondrium, and early in February there were all the signs of a deep-seated abscess below right ribs, while patient's general condition was such as to excite considerable alarm. Abscess was opened by Mr. Bickersteth with potassa fusa, integuments having been previously divided. Nearly a pint of 'tolerably healthy pus not mixed with bile escaped after a few days.' All hepatic symptoms ceased and she slowly recovered strength. Some weeks afterwards, another small superficial abscess was opened at umbilicus. The second opening soon closed, but first continued to discharge small quantities of pus and of a glairy fluid, and sometimes a little blood. This opening was situated about two inches above and to right of umbilicus, and at about an equal distance from natural situation of fundus of gall-bladder. It had a diameter of about 2 lines, was slightly depressed below surface, and was surrounded on all sides for about 2 in. by considerable induration of abdominal parietes, which, in an upward direction, amounted almost to a stony hardness. On introducing a probe the fistula seemed to take an upward direction, but as instrument caused pain and slight bleeding it could not be carried farther in than half an inch. Patient complained of dragging pains round opening and in right hypochondrium, and of occasional attacks of nausea and headache, but there were no other indications of constitutional disturbance. From the history I expressed the opinion at my first visit that abscess had not been in liver, but in gall-bladder, and I ventured to predict that sooner or later this would be proved by discharge of gall-stones through fistulous opening. Poultices were kept constantly applied over opening, and quinine with nitric acid, and occasionally purgatives, were ordered to be taken internally.

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[1] De sedibus et causis morborum, Epist. xxxvii.

[2] Op

On July 28 first gall-stone came away through opening. It was about size of a pea and presented several facets. Its passage through fistula was attended for several days by considerable pain, and its escape was followed by discharge of a good deal of thick yellow pus. On Aug. 15 three more concretions came away, one somewhat larger than first, two others very small. There was again for some hours a good deal of pain, and after exit of calculi a discharge of thick matter with a small quantity of blood. In September and October two or three more concretions escaped, and on Dec. 19, after two days and nights of intense pain, a polygonal calculus found its way out, which was fully half an inch in diameter. Another smaller concretion (ninth) escaped in Jan. 1868.

After this, induration surrounding opening greatly diminished, but there was still a mass of stony hardness, about size of a walnut, immediately above it. Opening was not more than a line in diameter and was retracted to bottom of a deep depression with puckering and induration of surrounding skin. This change was mainly due to greater thickness of abdominal wall from deposit of fat. Several times after escape of ninth stone, and in intervals of escape of previous ones, opening showed a tendency to close and caustic was applied. On one occasion an attempt was made to dilate fistula by a tent of sea-tangle, but removal of the swollen tent from tortuous tract caused so much pain that procedure was abandoned. Patient gained flesh and strength, looked extremely well, had a good appetite and digestion, and only suffered from inconvenience of discharge from fistulous opening.

Early in June 1868, another stone, tenth and last, came away. Opening continued to discharge thin pus, and it did not finally close till August 1869, but since then it has shown no sign of reopening, and there has been no pain nor induration in vicinity of cicatrix (1876).

On Sept. 27, 1868, more than three months after passage of last stone, and while fistulous opening was still discharging, patient was again suddenly seized with rigors, vomiting and fever, but this time associated with urinary symptoms—frequent micturition, intense burning pain in meatus urinarius, and presence of blood in small quantity, and subsequently of pus, in urine. These symptoms subsided after about a fortnight, but on Nov. 8 pain, vomiting, and fever returned in an aggravated form, and a very tender deep-seated swelling, about size of a small orange, could be felt in right groin half-way between crest of ilium and pubes. Urine was now perfectly limpid and free from pus or blood. Sir Henry Thompson, who saw patient with me in consultation, agreed in thinking that swelling was connected with right ureter. After a few days pus returned to urine, which also contained many crystals of lithic acid, but no stone. Tumour in right groin could still be felt on Nov. 30, but it was much smaller, and it soon entirely disappeared, and patient regained her usual health, urine, however, still containing a little pus. In August 1869, about time that fistula in abdominal parietes closed, pus disappeared from urine.

From this time patient remained in good health until Sept. 7, 1870, when she was again suddenly seized with rigors, vomiting, violent paroxysmal pain in region of right kidney stretching round to cicatrix and to right hip, much fever and frequent micturition, but urine contained no blood nor pus, and there was no induration nor swelling in neighbourhood of cicatrix. These symptoms continued in a more or less severe form till Sept. 26, when suddenly several ounces of pure pus were discharged with urine, and on following morning a calculus was passed from urethra. All severe symptoms at once subsided, and although urine for some weeks contained a good deal of pus, this at last disappeared and patient has till now (1876) enjoyed good health, except that she has suffered occasionally from aching and dragging pains in region of liver and right kidney.

Dimensions of stone were as follows:—Length $\frac{2}{5}$ inch, width $\frac{1}{5}$ inch, thickness, $\frac{3}{20}$ in. Its weight was 3 gr. It was analysed by Mr. Thomas Taylor, who found it to be composed of uric acid.

The following case occurred while I was pathologist to Middlesex Hospital, and was recorded by me in the Pathological Transactions (vol. xii. p. 85). The sequence of events was probably as follows:—

1. Ulceration of the interior of the gall-bladder from the presence of a gall-stone, perforation of its coats, and the formation of fistulous communications with the duodenum and colon, by which the calculus escaped into the bowel, as in the specimen in St. Bartholomew's Hospital Museum already referred to (p. 499).

2. Rupture by straining of some of the adhesions between the bowels, and the formation of a circumscribed fæcal abscess which opened externally.

3. Blood-poisoning, pyæmic abscesses, lobular pneumonia, and pericarditis.

Case CLXXI.—*Fistula in Abdominal Parietes opening into a circumscribed Cavity which communicated with Colon and Duodenum, and indirectly with Gall-bladder.*

B. Z——, aged 38, was admitted into Middlesex Hospital on Sept. 25, 1860, and died on Nov. 14. Married twice; eight children and six miscarriages.

About a year before death, without any apparent cause, she was suddenly seized with sickness, vomiting, and great prostration, which symptoms were followed by general fever and tenderness over abdomen. Bowels were regular. After a few weeks she recovered, and remained in her ordinary health up to Sept. 14, 1860.

On Sept. 13 she fancied that she strained abdominal muscles by carrying some heavy pails of water upstairs. On following morning she awoke with slight pain in abdomen, which was greatly increased after breakfast, and was then accompanied by sickness and vomiting of a green bitter fluid. She said pain was just as if her abdomen had been tied round with a rope. Sickness abated after three days, when bowels had been freely opened by medicine, but pain continued and patient became very weak. On admission into hospital, great tenderness of abdomen, which was most intense at umbilicus. Immediately to left of umbilicus was a superficial circular swelling with a firm dense rim and doughy in centre. Motions of bowels normal. Pulse 144. Great prostration.

Next day (26th) a little yellow pus of a stercoraceous odour, but exhibiting nothing except pus-cells under microscope, could be squeezed through umbilicus. After this date opening continued to discharge large quantities of fetid pus. From Sept. 30 to Oct. 17, pus was mixed with fæcal matter. On Nov. 9 opening ceased to discharge even pus.

About three weeks before death abscesses began to form in various parts of body, over right parotid, in soft parts of right hip, &c., and patient suffered from great dyspnœa and expectorated purulent sputa. It was impossible to examine chest, as slightest movement or manipulation caused great pain. The prostration gradually increased, and death took place on Nov. 14.

Autopsy.—Body greatly emaciated. An abscess containing seven or eight ounces of pus in right hip. At umbilicus was a fistulous opening large enough to admit a goose-quill. This opened into a sloughy cavity, size of a small orange, which communicated with transverse colon and duodenum, and indirectly with gall-bladder. Opening into colon was large enough to admit finger; colon at this place was much constricted, and its lining membrane injected and slightly ulcerated. Immediately to right of this opening gall-bladder was firmly adherent to colon. Gall-bladder small and contained about two drachms of whey-like fluid, without any tint of bile; cystic duct obliterated, but no gall-stones. Between fundus of gall-bladder and colon was a fistulous communication running somewhat obliquely, and just large enough to admit a No. 1 catheter; inner surface of gall-bladder around this opening marked by an extensive radiated cicatrix. The fistula between sloughy cavity and duodenum was large enough to admit a crow-quill, and opened into duodenum immediately beyond pylorus. Abdominal parietes around sloughy cavity were inseparably adherent to viscera; no fluid in peritoneum. About one pint of clear serous fluid in left pleural cavity. A few old adhesions over apex of left lung, which for most part was normal, but lower lobe contained several nodules of lobular pneumonia, largest about size of a walnut, grey, granular, bulging above surface on section and very friable. Two or three pints of turbid serous fluid containing numerous flakes of lymph in right pleural cavity, and lung glued to walls of chest anteriorly and at apex by recent lymph. Lower lobe of right lung collapsed, non-crepitant, sinking in water, smooth on section, and very tenacious.

Fully eight fluid ounces of a gelatinous, yellowish, opaque, puriform substance in pericardium, which could be scooped out in one semi-solid mass. This substance, on microscopic examination, was found to consist of fine fibrillated material with numerous lymph or pyoid corpuscles, but no true pus-cells with characteristic nuclei. Pericardium inseparably adherent to left ventricle over a space measuring 1½ in. in diameter. Outer surface of heart covered with membranous patches of lymph, many of which were firmly adherent.

CASE CLXXII.—*Biliary Fistula in Abdominal Parietes discharging bile.*

On Oct. 11, 1869, I saw with Mr. Curling a married lady, about 40 years of age, who had a biliary fistula. For many years she had been liable to sudden paroxysms of severe pain in right hypochondrium, accompanied by vomiting but never followed by jaundice. In March 1869 she first noticed a painful swelling in abdomen, below right ribs in front. Different opinions respecting this swelling had been expressed by different medical men who had been consulted. One was that it was a fibrous tumour, and another that it was a hydatid. The swelling increased, and as fluctuation became more distinct an opening was made into it in May, and many ounces of viscid, opaque, yellow fluid, without any trace of bile, came away. On June 3 a biliary concretion, not larger than a hemp-seed, was discharged through opening, and early in September four others, somewhat larger and with distinct facets, came away. On Sept. 18 she began to suffer from much pain about liver in front and stretching round to back, and after two or three days two other small concretions were passed and the pain was relieved. One week after this she awoke in night with agonising pain in right hypochondrium and back and violent retching. After a few hours these symptoms subsided, but two nights later (Sept. 27) they returned, and next morning she found her night-dress and the bedding saturated with bile. From that time until I saw her, fourteen days afterwards, there had been a steady discharge of dark green bile from the fistulous opening, which was situated half-way between umbilicus and lower edge of ribs in right nipple line. The fluid was discharged from opening at rate of from 1 to 2 oz. in hour, sometimes more and sometimes less. It was usually increased after a meal. It had all the characters of pure dark green bile. The patient was losing flesh and strength rather rapidly, and suffered much from pain and flatulence after meals. Urine was dark and contained bile-pigment, and motions were clay-coloured, with no vestige of bile, but there was scarcely any jaundice of skin or conjunctivæ. Three days after I saw the patient (Oct. 14) another small biliary concretion was discharged from opening; but there was no improvement in general symptoms, and patient continued getting weaker until about Nov. 7, when she had another attack of severe pain in right side and vomiting, and next day she found that discharge from fistulous opening had almost stopped and that there was plenty of bile in motions. The patient's general health gradually improved, and within a few weeks she was able to sail for the West Indies, but in Dec. 1872 there was still a minute fistulous opening below the right ribs discharging glairy mucus.

The several stages of this remarkable case appear to have been as follows:

1. A concretion, which had formed in the gall-bladder,

entered the cystic duct, causing paroxysms of hepatic pain and vomiting. It did not reach the common duct, and therefore there was no jaundice.

2. The cystic duct being closed, no bile could enter the gall-bladder; the bile already there was absorbed; the gall-bladder took on inflammation, and became distended with an opaque viscid fluid, forming a tumour which was appreciable through the abdominal parietes.

3. An opening was made into this cyst, and its contents evacuated. A fistulous opening remained, which discharged viscid fluid and gave exit to several small gall-stones.

4. With a fresh attack of biliary colic and vomiting the concretion in the cystic duct was dislodged and passed into the common duct, which it obstructed. The result was that the bile was prevented entering the bowel, and, passing along into the gall-bladder, escaped by the fistulous opening, and thus no jaundice resulted.

5. With another attack of biliary colic and vomiting the concretion escaped into the duodenum, the flow of bile was restored to its proper channel, and the fistulous opening closed.

This appears to be the only possible explanation of the facts of the case, but on this view it is extraordinary that a concretion which had blocked the cystic duct for many months should ultimately have been dislodged and passed into the common duct; and that the common duct should have become pervious after complete obstruction by a gall-stone for nearly six weeks was also almost more than, under the circumstances, there was reason to expect. The quantity of bile secreted by the liver in this case could not have been much under two pints in the twenty-four hours, and this though the patient was taking very little food.

I only saw her on one occasion, and she was much too ill to warrant any experiments with regard to the action of drugs on the secretion of bile.

Case CLXXIII. is another example of external biliary fistula produced by gall-stones. In addition to the three cases which I have now brought under your notice, I have met with two others, one in a lady aged 82, who never had jaundice; and a second in a lady upwards of 70, who had had two attacks of hepatic colic with jaundice, ten and five years before.

CASE CLXXIII.—*Numerous Gall-stones discharged by fistulous Openings at Umbilicus.*

Mrs. G——, 58, consulted me on April 4, 1872, by advice of Mr. J. C. Lynch of Sudbury. About three months before, she had been seized with vomiting and severe pain in region of liver. Very soon a hard painful swelling, about size of a hen's egg, appeared between right ribs and umbilicus, which after four or five weeks opened at umbilicus, discharging much pus, but no bile. Two days after a gall-stone came out, and since then many hundreds had been extruded, from size of a hemp-seed to that of a very large pea. She brought 328 with her in a parcel. The larger stones had caused much pain in passing. At time of her visit to me, patient had three fistulous openings surrounded by pouting red flesh and discharging a viscid fluid, just to right of umbilicus, and a stone had come away night before. For years she had suffered from 'bilious headaches,' but before this attack she never had pain in right side, and at no time jaundice.

July 25, 1876.—During the five months which followed Mrs. G.'s visit to me, 27 more gall-stones were passed. After this the fistula closed, and since then Mrs. G. has enjoyed better health than she had done for many years previously.

In the following case, although the patient had cancer of the liver, the chief pathological interest was the fact of a gall-stone having found its way into the interior of the portal vein.

CASE CLXXIV.—*Cancer of Liver—Ulceration of Gall-stone into Portal Vein—Phlebitis—Pyæmia—Peritonitis from rupture of hepatic abscess —Recent Endocarditis.*

Hannah L——, aged 57, adm. into St. Thomas's Hosp. Feb. 7, 1876. Father died at 45 of consumption; mother lived to 70; had 3 brothers and 7 sisters; none of them alive; all 3 brothers died of consumption. No history of gout, rheumatism, or syphilis. Throughout life had enjoyed good health, except that she had often suffered from severe spasms in abdomen after menstruation; catamenia had ceased at 40, but she had continued to suffer from severe darting pains in abdomen without vomiting or jaundice. Two years before admission into hospital she began to get weak and thin, and for eight months she had been subject to epigastric pain and had occasionally vomited her food. Still she continued to go about and earn her living as a laundress. Six weeks before admission, while washing, she was suddenly seized with severe pain in right little toe, which soon became swollen, red, and tender. In consequence of this she was obliged to give up work and kept her bed half the day.

On admission, patient was emaciated and had moderate jaundice of skin and conjunctivæ; there was gangrene, not spreading, of right little toe, with swelling, lividity, and some tenderness along outer side of right foot. But chief complaint was of pain in upper part of abdomen and back, constant, yet subject to severe exacerbations, persistent nausea and occasional retching. Tongue coated and dry down centre; bowels costive. No ascites. Liver apparently much enlarged, extending from level of nipple to below umbilicus; enlargement uniform; surface hard, uneven, and extremely tender. Pulse 96, weak; heart-sounds feeble; no bellows-murmur. No cough; lungs healthy. Temp. 101·2°. Urine 1015; no albumen.

After admission temperature soon fell to normal standard, but patient became rapidly weaker and died on Feb. 13.

Autopsy.—Liver attached by thick adhesions consisting in part of new growth to anterior abdominal wall and surrounding organs, in such a manner as to divide peritoneal cavity into two portions. Upper space contained a quantity of dirty greenish-yellow fluid, resembling bile mixed with pus. Upper surface of liver, which formed part of wall of this space, presented all the signs of recent acute inflammation and also two irregular softened patches with perforation, from which fluid exuded similar to that in cavity above. Anterior border of liver also adherent to pyloric extremity of stomach and to pancreas, a large irregular nodulated mass of cancer infiltrating and uniting them. This mass occupied situation of gall-bladder, and extended into portal fissure and adjoining portion of liver. Portal vein and common bile-duct passed into it; latter contained bile, and did not appear to be much obstructed. Portal vein, at about one inch from where it entered mass, opposite hilus of liver, was expanded into an irregular cavity with dark, ulcerated, sloughy walls, which contained an oblong gall-stone, measuring half an inch in longest diameter. No outlet to the cavity other than the branches of vein could be discovered. From this point branches of portal vein were intensely inflamed, and filled for some distance with adherent clot partially softened in centre; several of terminal branches also filled with adherent partially decolorised clot, and corresponding tracts of liver-tissue of a dead white or yellowish colour, and more or less softened, like pyæmic abscesses in an early stage, and surrounded by a zone of injection. Two large irregular tracts of this character on upper surface of right lobe had superimposed peritoneum sloughy, with several perforations from which contents had escaped into peritoneal cavity. There were also throughout liver, especially in left lobe, several nodules of new growth from ¼ to ¾ in. in diameter, hard, and where they approached surface flattened and slightly depressed. Spleen 7½ oz., soft; it contained several recent infarcti, partially softened. Kidneys small, with recent embolisms and deep scars from others of old date. Numerous small flattened nodules of new growth on under surface of diaphragm.

Lungs free from both infarcti and new growth. Valves of heart competent; but a small recent vegetation on centre of one of aortic flaps, and masses of recent vegetation on auricular surface of mitral valve.

C. ENLARGEMENTS OF THE GALL-BLADDER.

In the diagnosis of diseases of the liver it is important to keep in view the various causes of enlargement of the gall-bladder. Before closing this lecture I shall therefore say a few words on the distinctive characters of these enlargements, which, for clinical purposes, may be said to be due to five causes: viz.: I. Accumulation of Bile; II. Suppuration; III. Dropsy; IV. Gall-stones; V. Cancer.

I. *Enlargement of the Gall-bladder from Accumulation of Bile.*

Enlargement of gall-bladder from accumulation of bile, as I have already told you (pp. 161, 341), is one of the first consequences of obstruction of the common duct, and it is then distinguished by the following characters:—

1. Jaundice, which gradually becomes intense.
2. Absence of bile from the motions.
3. General enlargement and tenderness of the liver (see p. 161).
4. An elastic or fluctuating pear-shaped, somewhat tender tumour, projecting from the edge of the liver in the situation of the gall-bladder. Dr. Bright has recorded a case where the gall-bladder in this condition formed a fluctuating tumour extending almost to the crest of the ilium;[1] Dr. Babington relates a case in which the gall-bladder contained three wash-hand basins of bile;[2] and Copland mentions another in which it contained 8 pints of bile, and was so large as to protrude the false ribs on both sides.[3] It is not often, however, that the dimensions of the tumour are so great. The tumour may suddenly subside with the discharge of a large quantity of bile in the motions and the disappearance of the jaundice. When the obstruction of the bile-duct is permanent, the bile in the gall-bladder is often gradually absorbed and after a time a condition of atrophy may be substituted for that of dilatation. Sometimes the distended gall-bladder, when its coats are softened by inflammatory action or fatty degeneration, will rupture and cause fatal peritonitis, as in Case CLXV.

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[1] Abdom. Tumours. Syd. Soc. Ed. p. 271.

[2] Guy's Hosp. Rep. 1842-3, vol. vii.

[3] Dict. of Practical Med. vol. ii. p. 4.

cite ulceration of the mucous membrane or local peritonitis. The tumour may then become painful and adherent, and may increase in size.

V. *Enlargement of the Gall-bladder from Cancerous Deposit in its walls.*

Cancer of the gall-bladder is sometimes secondary to cancer of the liver or pancreas (Case CXXV. p. 380) or of some more distant organ (Case CLXXV.); more commonly the disease commences in the gall-bladder and the peritoneum or liver is affected secondarily (Case CLXXVI.). It is remarkable that in most of these cases the gall-bladder contains calculi, and the cancer appears to be the sequel of gall-stones (p. 372). Enlargement of the gall-bladder from cancer has the following characters.

1. There is a hard, sometimes nodulated tumour, about the size of an orange, more or less, in the region of the gall-bladder. Occasionally the tumour feels soft in the centre from softening of the cancerous matter, or from the cancer being chiefly at the neck while the fundus contains fluid (Case CLXXV.).

2. It is adherent and immovable.

3. It is very tender on pressure, and is usually the seat of severe lancinating pains.

4. Its growth may be rapid. Not unfrequently there is a previous history of biliary colic.

5. Jaundice and vomiting are common symptoms, owing to the extension of the cancer to the common bile-duct or to the pressure of the tumour on the pylorus.

6. Fistulous communications with the digestive canal, and particularly with the colon, are not uncommon, and consequently the passage of a large gall-stone, with or without hæmorrhage per anum, concurring with a tumour like that now described would corroborate rather than refute the diagnosis of cancer (see p. 499).

7. There is rapid emaciation with the usual phenomena of the cancerous cachexia.

Treatment of Enlargement of the Gall-bladder.

1. The treatment of over-distension of the gall-bladder with bile has been already considered under that of jaundice from obstruction of the bile-duct. (See p. 365.)

2. In the course of this lecture I have told you what mea-

sures you must have recourse to in inflammation of the gall-bladder. It now only remains for me to add:—

a. That in all such cases the patient must be cautioned against the risk of a severe muscular strain or a slight blow. Cases have been recorded where from such causes the gall-bladder has been ruptured and fatal peritonitis has been the result.[1]

b. That now and then it will be necessary to puncture the gall-bladder and evacuate its contents (see Case CLXX.), but that this ought never to be done except when the tumour is growing so rapidly that there is imminent danger of its bursting, or the constitution is being worn out by hectic fever. From what has been stated it is also obvious that the operation is rarely advisable when there is jaundice with absence of bile from the motions. If there be no adhesions over the tumour it will be necessary to produce them by means of caustic potash.

3. Accumulations of gall-stones in the gall-bladder must be treated in the manner already described (see p. 504); and lastly,

4. In cancerous enlargement all that can be done is to relieve distressing symptoms and promote euthanasia.

In conclusion, I may bring under your notice the two following cases of cancer of the gall-bladder, which came under my observation a few years ago and were reported by me in the eighth volume of the Pathological Transactions. In one case the cancer of the gall-bladder was secondary to cancer of the rectum and liver; in the other the cancer of the liver appeared secondary to that in the gall-bladder.

Case CLXXV.—*Cancer of Rectum—Secondary Cancer of Liver involving Gall-Bladder and obliterating Cystic Duct—Enlargement of Gall-bladder.*

A. B——, a female aged 53, was admitted into St. Mary's Hospital on Aug. 29, 1856. She had been suffering from pains in loins and abdomen for two months, and on admission she had also slight jaundice and constipation. Liver was not enlarged, but a tumour, size of a small orange, projected from lower border in site of gall-bladder. These symptoms increased, and a week after there was superadded uncontrollable vomiting. All treatment proved unavailing; jaundice became more marked, constipation more confirmed, and patient grew gradually weaker till death on Sept. 28.

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[1] There is a preparation showing this in the Museum of St. Bartholomew's Hospital, ser. xix. No. 14.

On post-mortem examination, small nodules of cancer scattered over peritoneal surface of intestines. A stricture of rectum from similar deposit commencing 1½ in. from anus and extending upwards for 3 in. Only portion of liver affected was lobus quadratus. In this there was a cancerous deposit, size of a small orange, which involved, and had obliterated, cystic duct. Coats of gall-bladder for one-fourth of their extent from duct were thickened by deposit; anterior three-fourths free from disease; whole gall-bladder was of a pale colour, and much distended, so as to project two inches in front of anterior margin of liver; it contained a milky flaky fluid, exhibiting under microscope numerous epithelial scales and two gall-stones about size of marbles. No trace of ulceration in any part of its lining membrane, and no adhesion between its outer surface and any of viscera. Bile in duodenum. Mucous membrane of stomach, the spleen, kidneys, and lungs free from disease.

Case CLXXVI.—*Destruction by Cancerous Ulceration of the Gall-bladder, and communication of resulting cavity with Transverse Colon—Cancer of Liver.*

S. P——, aged 56, a coach-painter, came under my care on Aug. 14, 1856. He stated that, when a young man, he had an attack of jaundice preceded by severe cramps in stomach. Fourteen years before I saw him he had suffered from rheumatic fever, followed by palpitations and other symptoms of cardiac disease. Father had lived to a great age, and mother had died at 86 of cancer of uterus. He was his mother's last child, and was born when she was nearly 50.

Three months before he applied to me for relief he began to suffer for first time from a pain in region of liver, but he continued regularly at work till middle of July. About this time he was seized with severe abdominal pains, vomiting, and purging. He continued at work, although irregularly, for a fortnight longer, but on 1st of August he was obliged to take to bed. On August 7 he became jaundiced. The following notes were taken when he was seen by me on August 14. 'Is very much emaciated; conjunctivæ are of a deep yellow colour, and countenance has an anxious cachectic aspect expressive of inward pain. Tongue has a yellowish fur; vomiting continues, almost everything he swallows being immediately rejected and sometimes apparently before it reaches stomach. Bowels relaxed. Two or three days ago, his wife states that he passed by stool a quantity of black matter like blood. Complains of sharp, shooting pain in hepatic region, coming on at intervals. Hepatic dulness in right mammary line extends four inches below margin of ribs, and a tumour can be felt in region of gall-bladder, 2 or 3 in. in diameter, immovable, apparently connected with liver, and very painful on pressure. Patient does not sleep on account of pain; pulse 100. A diastolic blowing murmur is heard over the middle of sternum.'

Was ordered milk-diet, wine, opiates, and various remedies to check vomiting, including naphtha and dilute hydrocyanic acid. Nothing, however, proved of any avail; he gradually sank, and died on Aug. 19. Previous to death stools had assumed a perfectly natural colour and consistence.

Autopsy.—Opaque patches on surface of heart and several small vegetations on both mitral and aortic valves. Lungs healthy.

No effusion in peritoneum; spleen and kidneys normal. Scattered throughout substance and over surface of liver were a number of small white masses of morbid deposit, varying in size from that of a pea to that of a small orange. Those on surface umbilicated in centre, and from all of them exuded on pressure a milky juice containing a multitude of 'cancer-cells' and free nuclei. These cells varied in size from $\frac{1}{320}$ to $\frac{1}{1500}$ of an inch, and in form were rounded, elliptical, fusiform, pear-shaped, &c., while their nuclei were large and well defined. Among them were a few 'mother-cells.' Transverse colon firmly adherent to anterior margin of liver, at a part corresponding to situation of gall-bladder; and on slitting up bowel, its interior was found to communicate by an opening as large as a half-penny piece with a cavity hollowed out in substance of liver, measuring 2½ in. from before backwards and 1½ in. from side to side. Walls of this cavity presented an irregular sloughy aspect, being composed of disintegrated hepatic and cancerous tissue, and interior filled with a dark-brown pultaceous fluid containing a piece of potato-skin and other débris of food. This cavity corresponded exactly in position to site of gall-bladder, no trace of walls of which could be seen; remains of obliterated cystic duct and artery were made out embedded in a mass of cancerous deposit, size of a Spanish chestnut, which also compressed, but did not obliterate, common hepatic duct. Margins of opening in transverse colon and whole circumference of corresponding portion of bowel thickened by deposit which narrowed calibre of gut, so as to produce a stricture barely admiting point of finger. Bowel above this stricture much dilated and exhibited on its mucous surface a number of superficial circular ulcers, largest being about size of a silver penny-piece. The continuation of gut beyond stricture was contracted. No disease of any other portion of intestines or of stomach, but pylorus was compressed by cancerous deposits in liver.

LECTURE XIV.

THE CROONIAN LECTURES ON FUNCTIONAL DERANGEMENTS OF THE LIVER.[1]

NOTICE OF DOCTOR CROONE—PRESENT NOTIONS AS TO FUNCTIONAL DERANGEMENTS OF LIVER UNSATISFACTORY—A. FUNCTIONS OF THE LIVER IN HEALTH. HISTORICAL SKETCH; GALEN'S VIEWS; OBSEQUIES OF LIVER BY BARTHOLIN; MODERN VIEWS; FUNCTIONS OF LIVER THREEFOLD. I. SANGUIFICATION AND NUTRITION. II. DISINTEGRATION OF ALBUMINOUS MATTER. III. SECRETION OF BILE; COMPOSITION, ORIGIN, QUANTITY, AND USES OF BILE. B. FUNCTIONAL DERANGEMENTS OF LIVER. OBJECTIONS TO EXISTING CLASSIFICATION. PROPOSED CLASSIFICATION. I. ABNORMAL NUTRITION. 1. CORPULENCE; 2. EMACIATION. a. DEFICIENCY OF BILE; b. DIABETES; c. OTHER VARIETIES OF EMACIATION. II. ABNORMAL ELIMINATION; SYMPTOMS OF RETAINED BILE; CHOLESTEARÆMIA.

MR. PRESIDENT, FELLOWS OF THE COLLEGE, AND GENTLEMEN,—It may interest some of you if, by way of preface, I say a few words respecting the founder of the course of lectures which I am honoured by delivering before you. For the particulars I am indebted to the 'Roll' of the College, edited by our learned colleague Dr. Munk.

Dr. William Croone was born in London and educated at Emanuel College, Cambridge. He became a Fellow of this College on July 29, 1675, and was Censor in 1679. In 1659 he was elected Professor of Rhetoric at Gresham College, and shortly afterwards he was made Secretary of the Royal Society, which then held its meetings in Gresham College. He resigned his professorship in 1670, on being appointed Lecturer on Anatomy at Surgeons' Hall. He died in 1684, and was buried in the churchyard of St. Mildred's in the Poultry. He left behind him a plan for two lectureships, which he had designed to found—one course of lectures to be read before the College of Physicians, after a sermon to be preached in the church of St. Mary-le-Bow; the other to be delivered yearly before the Royal Society, upon the nature and laws of muscular motion. His will contained no provision for the endowment of these lectures; but his widow (a daughter of Alderman Lorimer, who subsequently married Sir Edwin Sadleir, Bart.) carried out his intention by devising in her will the King's Head Tavern on Lambeth Hill, Knight Rider Street, in trust to her executors, to settle four parts out of five upon the College of Physicians for the purpose of founding the annual lectures now known as the Croonian Lectures, and the fifth part to found the Croonian Lecture of the Royal Society. A fine portrait of Dr. Croone was presented to the College in 1738 by Dr. Woodford, Regius Professor of Physic at Oxford, and is now suspended in the Censors' room.

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[1] Delivered before the Royal College of Physicians in 1874.

The founder of these lectures made no restriction as to their subject, as he did with regard to the lecture before the Royal Society; and it has been customary for each lecturer to select some subject in practical medicine which his experience has been most calculated to elucidate. The subject which I have chosen is one which it appears to me is well worthy of the attention of this College, and of medical men in general, viz., The Functional Derangements of the Liver. Professional opinion as to what constitutes functional disorder of the liver is vague and unsatisfactory. There is no expression more common among both patients and their doctors than that the 'liver is out of order,' or that certain symptoms are due to 'biliousness,' and yet few medical writers have undertaken to define with accuracy what symptoms are referable to a disordered liver. It is to be feared that symptoms are sometimes referred to the liver, with which it has little or no concern; while, on the other hand, there are grounds for suspecting that many symptoms, at first sight apparently referable to other organs, and even grave degenerations of tissue and organic disease, not only of the liver itself, but throughout the body, may be traced back to functional derangements of the liver, although some of these may as yet be imperfectly understood. It is remarkable how systematic writers on Medicine and on Diseases of the Liver in particular entirely ignore the subject of functional disorders of the liver. The remarks which follow must be regarded as a feeble attempt to sketch, with the light of recent investigation, those symptoms and morbid conditions which may fairly be put down to a disordered liver. They do not pretend to place the subject upon a firm and lasting basis; but, by calling attention to its importance and provoking discussion, it is hoped that they will prove a stepping-stone to a more certain knowledge of it, and in the meantime that they will help to supply what appears to be a deficiency in medical literature.

A. FUNCTIONS OF THE LIVER IN HEALTH.

Before proceeding to discuss the results of derangement of the liver, it will be necessary for me to refer at some length to the functions of the organ in its healthy state. As in the case of the pathology of pyrexia and of inflammation, so with regard to the healthy functions of the liver, it is not a little remarkable that modern investigations have tended to reproduce in a scientific form certain crude opinions entertained by the earliest writers on Medicine. From its large size, and from the extensive system of blood-vessels connected with it, the liver was believed by the Fathers of Medical Science to be the seat of many most important functions, and to be, in fact, the central organ of vegetative life. Galen, for example, taught that the liver was the centre of animal heat, that it was the seat of sanguification, and that it was the starting point of the venous system. He assigned to the veins distributed over the intestines the function of imbibing the fluid nutriment and of conveying it by the vena portæ to the liver, where he supposed that the processes of sanguification and of the generation of animal heat took place. He then traced the passage of the blood through the hepatic veins to the heart, and hence he regarded the liver as the starting point of the venous system. For upwards of sixteen centuries these views of Galen—more or less modified—were generally accepted by physiologists and physicians, and as late as the seventeenth century they were in the main upheld by our own Harvey. But the discovery in the first half of the seventeenth century of the lacteals and thoracic duct showed that chyle was conveyed to the blood independently of the portal vein and of the liver. The result was that this organ at once fell from its high estate, and ceased to be regarded as serving any purpose in sanguification. Thomas Bartholin, in his 'Defence of the Lacteals and Lymphatics against Riolanus,' wrote for the liver an epitaph, in which the end of its dominion was announced, and its function was declared to be henceforth limited to the secretion of bile.

Vivit, floretque pro bile separandâ, sed, si sanguinem conficiendum spectemus, funeratum creditur. Ivimus illi exsequias, nunquam redituro. Nam . . . .

. . . . Facilis descensus Averni,
Sed revocare gradum, superasque evadere ad auras
Hoc opus, hic labor.[1]

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[1] Defensio vasorum lacteorum et lymphaticorum adversus J. Riolanum. Hafniæ 1655, p. 8.

Although it was *à priori* improbable that the largest gland in the body, deriving large supplies of blood from different sources as well as holding peculiar relations to the blood returning from the placenta in the fœtus and from the stomach and intestines in the adult, should have as its sole function the secretion of a fluid which is apparently of less importance in digestion than the gastric or pancreatic juice, yet for nearly two centuries the only object of the liver was believed to be the secretion of bile: and down to the present day its functional derangements are constantly spoken of as restricted to the secretion of bile abnormal in quantity or in quality. For example, our late learned colleague Dr. Copland, one of the few modern medical authors who have discussed the functional disorders of the liver, describes them as coming under three heads, viz.: 1. Diminished secretion of bile; 2. Increased secretion of bile; and 3. Secretion of morbid or altered bile;[1] and this classification probably represents with tolerable accuracy the views of the great majority of modern practitioners of medicine. It is the belief that the sole function of the liver is the secretion of bile which has given rise to the expression in common use by professional men as well as laymen, that the 'liver will not act,' when all that is implied is a constipated state of the bowels. But the physiological investigations made within the last quarter of a century have in a great measure restored the liver to its former place of importance in the animal economy; they have shown that the secretion and excretion of bile are far from being the most, if they be not the least, important of its functions; and they have consequently added to the number of its functional derangements.

I. In the first place it is now known that the liver is one of the organs mainly concerned in the process of sanguification. So long ago as 1820, it was shown by Magendie and Tiedemann that the absorption of nutritive matters from the bowel was not limited to the lacteals, but that part was taken up into the blood through the portal vein;[2] and the researches of subsequent physiologists have clearly established that the liver exercises most important functions in assimilation and nutrition. The most valuable contributions towards our knowledge of this matter have been the researches of Claude Bernard and other observers, who have shown that the liver has the power of making and storing up for a time within its cells glycogen ($C^6H^{10}O^5$), a substance resembling dextrin ($C^6H^{10}O^5$) in its chemical composition and reactions, and like it capable of conversion into sugar by the action of albuminoid ferments. This substance always exists in the liver in larger amount during digestion than during fasting, attaining its maximum usually about four or five hours after a meal. It does not yet seem certain what the materials are from which it is mainly formed, but there can be no doubt that its amount is increased by the use of starchy or saccharine food. The starch ($C^6H^{10}O^5$) of the food is believed to be converted into grape-sugar or glucose ($C^6H^{12}O^6$) by the saliva and pancreatic secretion, while the cane-sugar ($C^{12}H^{22}O^{11}$) is transformed by the intestinal secretion into grape-sugar and another form of sugar called lævulose ($C^6H^{12}O^6$). The glucose and lævulose are absorbed by the intestinal veins and carried by the portal vein to the liver, where they are converted partly perhaps into fat, but mainly into glycogen, which is stored up in the hepatic cells, and distributed for the nutrition of the tissues during the intervals of fasting. The circumstance, however, of glycogen being formed in considerable quantity in the livers of animals who have been fed for a month or more on flesh alone, and the fact that its quantity in the liver of a dog is always increased after a meal of flesh, show that it can also be produced from albuminous matter. The albumen of the food is converted by the gastric juice into peptone, which is also absorbed by the intestinal veins and carried to the liver, where it is believed to be decomposed into glycogen and nitrogenous products such as leucin ($C^6H^{13}NO^2$) and tyrosin ($C^9H^{11}NO^3$), which are ultimately resolved into urea (CH^4N^2O).[1] The glycogen derived from these two sources does not remain long in the liver, for the large quantity formed after a meal is quickly diminished on fasting. It is not got rid of by the bile-ducts, for bile contains neither glycogen nor sugar. But either as glycogen, or more probably as sugar,[2] into which it is believed by Bernard and most other physiologists to be reconverted through the action of an albuminoid ferment in the liver or in the blood, or transformed in some other way, it enters the blood by the hepatic veins.

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[1] Medical Dictionary, ii. 723.

[2] Versuche über die Wege auf welchen Substanzen aus dem Magen und Darm-Canal im Blut gelangen. Heidelberg, 1820.

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[1] See Fick in Pflüger's Archiv, vol. iv. p. 40; also Schultzen and Nencki, Zeitschrift für Biologie, vol. viii. p. 124.

[2] In reference to the difference of opinion still existing on this matter, Dr. Brunton writes as follows, in Sanderson's Handbook for the Physiological Laboratory 1873, p. 508: 'While Bernard considers that the formation of sugar goes on in the liver constantly during life, this has been denied by Pavy, Ritter, Meissner, and Schiff, who hold that it only occurs after death, or under pathological conditions, such as disturbance of the respiration or circulation during life. They base their opinions

One object of the glycogenic function of the liver is supposed to be that of continuously supplying an easily oxydisable material, such as sugar, which, in the presence of oxygen and albuminous matter, is readily converted in the lungs into carbonic acid and water and thus contributes to the maintenance of animal heat. But although there is still some difference of opinion among those most competent to judge, the bulk of evidence goes to show that a portion only of the glycogen formed in the liver is transformed into sugar to be burnt in the lungs, and that the maintenance of animal heat is far from being its chief use. There are good grounds for believing that it assists in cell-growth; for, just as in plants the presence of sugar seems to be necessary for the most rapid development of cells, so in animals glycogen can be found wherever cell-growth is actively going on. Bernard and Rouget have found it in abundance in the cells of the placenta and amnion; and Rouget in many fœtal tissues, such as cartilage, muscle, and the epithelial cells of the skin;[1] and it is also present in the inflammatory products of pneumonia, which are mainly made up of leucocytes, and in new growths whenever cell-formation is active. Hoppe-Seyler has also shown that it is an ingredient of colourless blood-corpuscles, so long as they are active, but that when they lose their power of motion the glycogen disappears and is replaced by sugar.[2] In connection

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on the observations that the liver contains little or no sugar when examined immediately after death, and that the blood of the hepatic vein does not contain more sugar than that of the portal or jugular veins. It is quite true that sugar is found in very small amount in fresh livers; but the smallness of the quantity is in all probability due to the constant circulation through the liver during life washing the sugar out of it as soon as it is formed (Flint). The statement that the blood of the portal contains as much sugar as that of the hepatic vein rests on experiments vitiated by the omission to place a ligature on the former while removing the liver, so that, the hepatic vein having no valves, the blood from it flowed back into the portal system. When this fallacy is avoided, sugar is found in much larger proportion in the hepatic than in the portal vein. To meet the objection that sugar thus found has been formed after death, blood has been taken from the right side of the heart, or vena cava, and the quantity of sugar it contained compared with a similar specimen of blood from the jugular vein. Every precaution was taken to avoid disturbance of the circulation, yet the sugar in the former was found to exceed that in the latter considerably (Lusk).'

[1] Journal de Physiologie, 1859, tome ii.

[2] Med. Chem. Untersuch. 1871, p. 486.

with these observations, it is important to note that the blood, on emerging from the liver, is much denser and contains a far larger proportion of solid constituents (although less fibrin), and is also far richer in white blood-corpuscles, than the blood before it enters the liver. Bernard, Lehmann, and McDonnell ascertained that in blood drawn from the hepatic vein, the colourless corpuscles are from five to ten times more numerous than in blood taken from the portal vein;[1] while Hirt of Zittau estimated that the proportion of the colourless to the red corpuscles was in the portal venous blood as 1 to 524, but in the hepatic as 1 to 136.[2] The red corpuscles also from the hepatic vein are said to have a sharper outline and less tendency to aggregate into rolls, and to dissolve less readily in water than those from the portal vein. Again, we have the remarkable observations of Weber, confirmed by Kölliker, respecting the extensive generation of blood-corpuscles in the liver of the embryo. In the early stages of fœtal life the blood-cells multiply throughout the entire mass of the blood; but when the liver begins to be formed this process ceases, and a very active formation of colourless blood-cells is set up in the liver, these colourless cells undergoing a gradual change by the development of colouring matter in their interior into red corpuscles. According to Kölliker, this new formation of blood-corpuscles in the liver continues during the whole of the fœtal life of mammalia.[3] The observations quoted above make it probable that the liver in the adult continues to perform the functions which pertain to many different tissues of the fœtus, the glycogen secreted in its cells combining with nitrogen and forming an azotised protoplasm, which maintains the nutrition of the blood and tissues. Dr. R. McDonnell has suggested that part of the glycogen of the liver combines with nitrogen furnished by the fibrin of the blood, which is disintegrated in its passage through the liver, and that the result is a new protein substance which enters the circulation.[4] Our colleague Dr. Pavy is also of opinion that glycogen is capable of transformation into fat. As he contends, it is beyond dispute that starch and sugar introduced with the food lead in the animal system to the production of fat, while his experiments have shown that the ingestion of these principles is followed by a marked increase in the amount of glycogen in the liver.[1] The production of glycogen, then, may be regarded as the first step in the assimilation of the starchy and saccharine elements of our food: and, as these elements are known to proceed on to fat, glycogen would seem to occupy a position intermediate between the two. The process of assimilation may go on to the production of fat in the liver, or it may stop short at the formation of another principle, which escapes from the liver and is elsewhere transformed into fat. Lastly, there are good grounds for thinking that both glycogen and sugar serve some purpose in muscular action; at all events, it has been found that the quantity of sugar in blood becomes greatly diminished in passing through the vessels of contracting muscles.[2] According to Bernard, this destruction of sugar in muscle is due to lactic fermentation.[3]

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[1] See McDonnell's Observations on the Functions of the Liver. Dublin, 1865.

[2] Müller's Archiv, 1856; and Carpenter's Principles of Human Physiology, 7th edition, p. 228.

[3] Todd and Bowman's Physiology, 1856, ii. 263; Carpenter's Principles of Human Physiology, 7th edition, 1869, p. 214; and Kölliker's Manual of Human Histology, Sydenham Society edition, 1854, vol. ii. p. 342.

[4] Op. cit.

There may be other ways in which the liver contributes to assimilation and the nutrition of the body; but enough has been said to justify us, notwithstanding the prediction of Bartholin, in restoring to the organ the important function claimed for it by Galen and his successors, viz., that of sanguification.

II. But, in the second place, modern research has made it probable that the liver is endowed with a function not suspected by Galen, and which, from a pathological point of view, is even more important than that which we have been considering. Many observations, pathological as well as physiological, point to the conclusion that the liver is not only a blood-forming, but a blood-destroying or purifying organ, and that it contributes in a great degree to the destruction of albuminous matter derived from the food and textures, and the formation of urea and lithic acid, which are subsequently eliminated by the kidneys. First, there seems to be little doubt that the albumen and fibrin of the blood become largely disintegrated in the liver. Lehmann and Bernard have shown that, while portal blood contains much fibrin, blood from the hepatic vein contains little or none.[1] Brown-Séquard has calculated that no less a quantity than 2,690 grammes, or about 86½ oz., of fibrin is daily lost to the blood in its passage through the digestive organs and the liver.[2] If this be so, we can readily understand that, when anything occurs to interfere with this fibrin-destroying function, there should be a rapid increase of fibrin in the blood, as we know to occur in acute rheumatism and in other diseased states. Moreover, there are grounds for believing that, while white blood-corpuscles take their origin in the liver, the red corpuscles are destroyed there, and that the nitrogenous colouring matters of the urine are partly the result of this destructive process. Gréhaut has ascertained that there is a positive destruction of hæmoglobin in the passage of blood through the liver.[3] Red blood-corpuscles are known to be at once destroyed when brought in contact with a solution of bile-acids of a certain strength (12 per cent., Legg),[4] while the researches of our late colleague Dr. Bence Jones make it very probable that the various shades of yellow, brown, and pink presented by the sediments of the urine are due to different degrees of oxidation of the pigment of the bile.[5] But, secondly, there is evidence that the liver is largely concerned in the formation of the nitrogenous matters which are eliminated by the kidneys.

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[1] F. W. Pavy, The Nature and Treatment of Diabetes, 2nd ed. 1869, p. 113.

[2] Sanderson's Handbook for the Physiological Laboratory, 1873, p. 508.

[3] Lectures delivered at the College of France, London Medical Record, October and November, 1873.

1. First, there is the well-known fact, to which I shall have occasion to refer again in more detail, that among the most constant signs of functional derangement of the liver is an imperfect formation of urea, evidenced by the deposit of lithic acid or lithates and of a dark colouring matter closely allied to lithic acid in the urine.

2. Secondly, when a great part of the liver has been destroyed by disease, the urea discharged in the urine becomes greatly lessened, or it entirely disappears. For example, when a great part of the liver has been destroyed by cancer, there

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[1] McDonnell, op. cit. p. 29; G. Budd, Diseases of the Liver, 3rd ed. 1857, p. 47.

[2] Journal de Physiologie, i. 304.

[3] Sanderson, op. cit. p. 498.

[4] Kuhne, Archiv für path. Anat. 1858, Bd. xiv. p. 324; Robin, Mémoires lus à la Société de Biologie pendant l'Année 1857; and Dr. J. W. Legg, Bartholomew's Hospital Reports, vol. ix. 1873.

[5] G. Budd, op. cit. p. 34; Sanderson, op. cit. p. 499. 'The very close resemblance of urine-pigment to bilifulvin is strongly suggestive of an hepatic origin. . . . An argument in favour of a liver origin may perhaps be drawn from the effect of liver-diseases on the urinary pigment.' (Parkes, On the Urine, 1860, p. 30.)

has been found to be a remarkable diminution of urea.[1] Thirty years ago, our colleague Dr. Parkes examined the urine in a number of cases of hepatitis and hepatic abscess in India, and found that in some instances there was abundance of urea, and in others scarcely any, while in some it was altogether wanting. The cause of the difference appeared to be the amount of suppuration. When this was excessive, so that the secreting substance of the liver was almost entirely destroyed, the amount of urea was greatly lessened, and in a degree proportioned to the extent to which the glandular tissue was destroyed by the abscess; and, on the contrary, when the liver was not suppurating, but was actively congested and enlarged, so that there was an increased activity of the secreting cells, the amount both of urea and lithic acid was increased.[2] Again, in that singular malady, acute atrophy of the liver, where every secreting cell of the liver becomes rapidly disintegrated, all trace of urea may disappear from the urine, its place being taken by albuminoid substances less oxidised, such as leucin and tyrosin, which are also found in large quantity in the hepatic tissue, as if they marked the arrest or modification of the transformation of albumen.[3] Lastly, there are grounds, to be referred to presently, for believing that, when cerebral symptoms supervene in any case of protracted jaundice with destruction of the liver, they are not due, as has been commonly thought, to saturation of the system with bile, but to non-elimination of urea. Recent observations confirm in a remarkable manner the conclusions to be drawn from what I have just stated. M. Genevoix, in a recently published thesis,[4] from observations of his own, as well as from those of MM. Charcot, Bouchardat, and others, concludes that disorders of the liver which do not seriously implicate its secreting tissue, such as congestion and some forms of jaundice, increase the amount of urea excreted, while the graver disorders, such as cancer, cirrhosis, and acute atrophy, diminish it very considerably. Precisely the same conclusion is arrived at by M. P. Brouardel in an elaborate memoir published in the 'Archives de Physiologie' for August and December, 1876. Ample evidence is there adduced to show that the quantity of urea voided in the urine in twenty-four hours depends upon; 1. the activity, more or less, of the hepatic circulation; and 2. the integrity or destruction of the secreting cells of the liver, the quantity being always greatly diminished in diseases of the liver which entail a great destruction of its secreting tissue, such as acute atrophy, cirrhosis, fatty liver, &c. From these observations, it is clear, as Meissner has argued, that 'withering and destruction of the liver-tissue is connected with an important diminution in the formation of urea,'[1] and that the quantity of urea voided in the urine furnishes most important evidence for grounding a prognosis in many hepatic disorders.

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[1] Parkes, On the Urine, 1860, p. 330.

[2] On the Dysentery and Hepatitis of India, by E. A. Parkes, 1846.

[3] Frerichs, Klinik der Leberkrankheiten, New Sydenham Society's translation, vol. i. p. 221; Murchison, Clinical Lectures on Diseases of the Liver, 1868, p. 229.

[4] Chez Delahaye, Paris, 1876.

3. There is experimental evidence that urea exists in large quantity in the liver, and that it is formed there. Dr. Parkes, in the Croonian Lectures for 1871,[2] informed us that the experiments of Heynsius and Stokvis, followed up by those of Meissner, Bullard, Perls, and others, had placed on a certain experimental basis the fact, that urea can be largely found in the liver; while the more recent observations of Cyon seem to prove that there is an actual production of urea in the liver. Meissner discovered large quantities of uric acid in the livers of birds, and of urea in the livers of dogs and cats. Cyon ascertained, on analysing blood obtained by introducing tubes into the portal and hepatic veins of dogs, that the blood from the latter vessels always contained much more urea than that from the former. In one experiment, the blood from the portal vein contained only ·08 grm. of urea in 100 cubic centimetres; but, after passing through the liver once, it contained ·14 grm.; and, after passing through the liver four times, 0·176 grm. He satisfied himself also that this increase was not due merely to washing out the liver, but that there was an actual formation of urea.[3] It is important to add, that the formation of urea in the liver is always greatly increased after food. Lithic acid has also been found repeatedly in the liver of man and mammalia, but always in small quantity; whereas, in birds, in which lithic acid takes the place of urea as the great eliminator of nitrogen, it also takes the place of urea in the liver, as it probably also does in the human liver under certain pathological conditions.

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[1] Henle's Zeitsch. für rationelle Medicin, Bd. xxxi. p. 246.

[2] Lancet, 1871, vol. i. p. 469.

[3] Centralblatt für die Med. Wissenschaften, August 1870, p. 580.

All these observations point to the liver as being largely concerned in the destructive metamorphosis of albuminoid matter, the products of which are eliminated by the kidneys,[1] although it is not improbable that other glandular organs, and even the corpuscles in the circulating blood, as believed by H. Ludwig and Fuhrer,[2] may contribute to the process.

As might have been expected, these oxydising and disintegrative processes, as well as those connected with the formation of bile, are attended by a production of heat. The average temperature of the body generally being between 98° and 99° Fahr., the temperature of the healthy liver reaches 104°, or even, according to Bernard, sometimes 106°.[3] Bernard has also shown that in dogs the temperature of the blood in the hepatic veins is considerably higher than that of the blood of the portal vein, and that the temperature of the upper part of the vena cava is higher than that of any other part of the body. This high temperature is no doubt due to the active chemical changes going on in the liver; and, as heat is absorbed during organisation and given off during disintegration, the high temperature generated in the liver makes it probable that the disintegrative processes taking place in the gland are in excess of the formative. On the other hand, when the activity of the chemical changes in the liver is impaired, as after ligature of the common duct or in jaundice from obstruction, the temperature of the body is often subnormal (see p. 319), and in animals there is no longer found an increased temperature of the blood in the hepatic veins. It follows, therefore, that the precise observation of modern times has confirmed the statement enunciated centuries ago by Galen, that the liver is a great centre of animal heat.

III. The third function of the liver is the secretion of bile. The composition of this substance is complex, and its uses are not yet sufficiently known. Human bile, as found after death, is usually a dark brown fluid, of tenacious consistence from the presence of mucin, which it derives from the gall-bladder and bile-ducts; but, when fresh, as it flows from the liver, it is a thin transparent liquid, of a golden yellow colour like that of yolk of egg, of a very bitter taste, of alkaline reaction, and having a specific gravity of about 1018. It has an unctuous feel and mixes freely with oil or fat. It contains from 9 to 17 per cent. of solid matters (the proportion being always greater soon after a meal), consisting for the most part of substances peculiar to bile. Excluding the mucin, its principal ingredients are—1. Bile-pigment; 2. Biliary acids combined with soda; 3. Cholesterin and fats, 4. Mineral matter, such as phosphates of soda, potash, lime, magnesia, and iron, chloride of sodium, and traces of copper.

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[1] In jaundice from obstruction of the bile-duct we have found that the nutritive functions of the liver are impaired or arrested, but it does not follow that there should be a corresponding impairment of the destructive functions of the organ. The fact, therefore, that in jaundice from obstruction the elimination of urea by the kidneys is in the first instance but slightly diminished, or even increased, is no argument against the liver being concerned in its production. If the jaundice be sufficiently protracted, non-elimination of urea and symptoms of blood-poisoning are not uncommon.

[2] Parkes, Lancet, 1871, vol. i. p. 470.

[3] On the Heat of the Body. The Gulstonian Lectures for 1871. By Dr. S. Gee, British Medical Journal, 1871, vol. i. p. 330.

The following analysis of bile obtained from a man aged 22, killed by an injury, was made by Frerichs:—

Water	859·2
Solid residue	140·8
Glycocholate of soda Taurocholate of soda	91·4
Fat	9·2
Cholesterin	2·6
Bile-pigment and mucus (of which, mucus about 1·4)	29·8
Salts	7·7

The yellow pigment is designated *bilirubin* ($C^{16}H^{18}N^{2}O^{3}$). On standing, it becomes greenish from oxydation and is converted into *biliverdin* ($C^{16}H^{20}N^{2}O^{5}$), which accounts for the dark colour usually presented by the bile in the gall-bladder after death and in the fæces. Biliverdin is also the principal colouring matter of the bile of the herbivora. Bilirubin is now known to be formed from blood-pigment or hæmoglobin by the hepatic cells in the passage of the blood through the liver. That this was the source of the bile-pigment was suggested at the end of last century by a distinguished Fellow of this College, Dr. W. Saunders, who observed: 'Green and bitter bile, being in common to all animals with red blood, and found only in such, makes it probable that there is some relative connection between this fluid and the colouring matter of the blood, by the red particles contributing more especially to its formation.'[1] This view, revived in our own day by Virchow, is supported by the apparent identity of bile-pigment with the pigment hæmatoidin found in old extravasations of blood, and by the fact that what appears to be bile-pigment can be produced from blood-pigment by the action of chemical reagents;[1] by the discovery of Zenker and Frerichs of crystals of hæmatoidin in inspissated bile and in the bile of jaundiced urine;[2] by the observation of Gubler that bilirubin and hæmatin give the same play of colours with nitric acid, except that the green colour is most persistent in the former, and the violet in the latter;[3] by the discovery of Frerichs, Kühne, and others, that when any substance such as bile-acids or even water, which has the property of dissolving blood-corpuscles and liberating hæmoglobin, is injected into the veins, bile-pigment (bilirubin) appears in the urine; and by the statement of Gréhaut, that there is a positive destruction of hæmoglobin in the passage of blood through the liver.[4] There are, on the other hand, grounds for believing that bile-pigment is in its turn converted into urinary pigment.[5] A substance presenting spectroscopic characters similar to those of urinary pigment can be prepared by deoxydation from bilirubin; and it is believed that in the organism bile-pigments are reduced by hydrogen or other reducing agents present in the intestines.[6] It is also a well-known clinical fact, that nothing influences so much the characters of the urinary pigment as functional or structural disease of the liver. The liver then, together with that osmotic circulation constantly going on between it, the blood, and the contents of the intestines, to be referred to presently, appears to be the medium of conversion of blood-pigment into bile-pigment, and of bile-pigment into urinary-pigment.

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[1] Treatise on the Structure and Diseases of the Liver, 3rd edition, 1803, p. 147.

The bile-acids in human bile are two—glycocholic acid ($C^{26}H^{43}NO^{6}$) and taurocholic acid ($C^{26}H^{45}NO^{7}S$). Both acids are derivatives of albumen and contain nitrogen; and taurocholic acid, to which the bitter taste of bile is due, contains all the sulphur of the bile. Both are in bile combined with soda, and both are what are called conjugate acids; that is to say, they are composed of cholic acid ($C^{24}H^{40}O^{5}$), which contains neither nitrogen nor sulphur, in combination with taurin ($C^2H^7NO^3S$), which contains both nitrogen and sulphur, and glycocin ($C^2H^5NO^2$), which contains nitrogen, but no sulphur.

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[1] Virchow's Cellular Pathology, English translation, p. 144; Kuhne, Lehrbuch der Physiol. Chemie, Leipzig, 1866, p. 89.

[2] Jahresb. von der Gesellschaft für Natur- und Heil-kunde in Dresden, 1858, p. 53.

[3] Gaz. Méd de Paris, 1859, p. 469.

[4] Sanderson, op. cit. p. 498.

[5] Bence Jones, referred to by G. Budd, op. cit. p. 34; Parkes, On the Urine, 1860, p. 30.

[6] Sanderson, op. cit. p. 499.

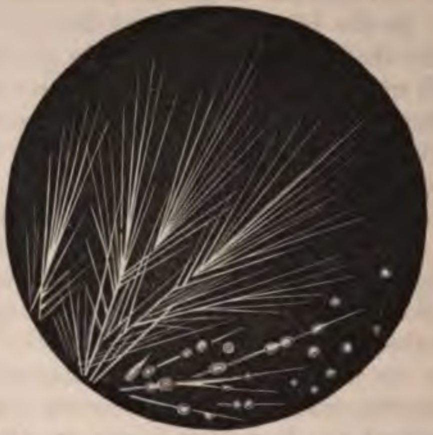

FIG. 32. Glycocholate of soda from ox bile after two days' crystallisation. At the lower part of the figure the crystals are melting into drops from the evaporation of the ether and absorption of moisture. After J. C. Dalton.

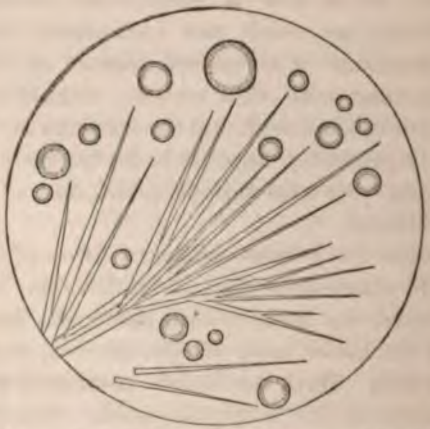

FIG. 33. Glycocholate and taurocholate of soda from ox bile after six days' crystallisation. The glycocholate is crystallised; the taurocholate is in fluid drops. After J. C. Dalton.

Cholesterin ($C^{26}H^{44}O$) is a fatty substance, which crystallises in colourless rhombic plates, one corner of which is often indented. It is met with in nerve-matter, in the spleen, in blood, and in certain morbid exudations, as well as in bile. An American physician, Dr. Austin Flint, jun., has endeavoured to show that cholesterin is formed for the most part, if not entirely, from nerve-tissue, from which it is taken up by the blood; and that one of the chief functions of the liver is to eliminate this cholesterin, the accumulation of which in the blood, from the liver ceasing to act, is attended by cerebral and other symptoms of blood-poisoning.[1]

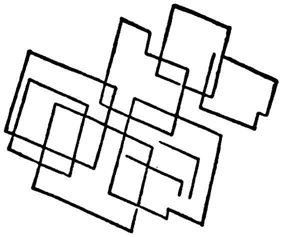

FIG. 34.—Crystalline Plates of Cholesterin.

Bile thus constituted is being constantly secreted by the liver. There is still much difference of opinion as to the part played by the liver in the formation of bile. It is generally admitted that the biliary acids are formed in and by the liver: but many physiologists and physicians still maintain that the bile-pigment is preformed in the blood and is merely separated from the blood by the liver; and they explain those cases of jaundice in which there is no obstruction of the bile-duct, by saying that bile-pigment accumulates in the blood from the liver ceasing to act, or from its function being suppressed.[2] But it has long appeared to me that there are weighty objections to this view. (See Lecture IX., p. 327.)

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The supposition that bile-pigment is formed in the blood appears to me for these reasons to be untenable.

The quantity of bile secreted by the liver has been shown experimentally to increase suddenly after a meal, reach its maximum in about two hours, and then gradually decline, while by abstinence it is greatly lessened. In considering the functional derangements of the liver, it is very necessary to remember that the total quantity of bile secreted in twenty-four hours by a man eating an ordinary amount of food is much larger than might be expected from that which is discharged from the bowel. The daily quantity of human bile has been usually calculated from what has been observed in dogs with artificial biliary fistulæ,[1] and the principal results have been as follow. According to Kölliker and Müller, a dog consuming daily about one-fifteenth of its own weight of meat secretes in twenty-four hours 36·1 parts of fluid bile in 1,000 parts of its own weight. According to Dr. George Scott, a dog consuming daily about one-fourteenth of its own weight of meat secretes in twenty-four hours 23·13 parts of fluid bile in 1,000 parts of its own weight. According to Bidder and Schmidt, a dog consuming daily about one-seventeenth of its own weight of meat secretes in twenty-four hours 19·19 parts of fluid bile in 1,000 parts of its own weight. Making allowance for the greater relative weight of the liver in the dog than in man,[2] it follows from these results that the amount of fluid bile secreted in twenty-four hours by a man weighing 160 lbs. and on full diet, is, according to

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[1] Recherches Expér. sur une nouvelle fonction du Foie. Paris, 1868.

[2] Budd, op. cit.; and G. Harley, Jaundice, its Pathology and Treatment. London, 1863.

Kölliker and Müller	66·742 oz.
Scott	42·763 „
Bidder and Schmidt	35·476 „

Similar experiments have been made on dogs by Nasse, Platner, and Stackman, and from their data Carpenter has calculated that a man weighing 154 lbs. should secrete daily about 40 oz. of bile. It may be assumed, then, from experiments on the lower animals, that the quantity of bile secreted in twenty-four hours by the human liver is about 40 oz.; and this inference is, on the whole, confirmed by what is observed in those rare cases where a biliary fistula discharging bile is produced in the human subject by ulcerative perforation of the fundus of the gall-bladder, the cystic duct remaining patent, while the common bile-duct is closed; although in some instances the quantity of bile has been less than in health, from the patient being greatly emaciated and taking little food. One case of the nature referred to has come under my own notice, the particulars of which are worth mentioning. The patient was a lady aged 40, whom I saw in October 1869, in consultation with Mr. Curling (see Case CLXXII., p. 519). Owing to an obstruction of the cystic duct by a gall-stone, the gall-bladder became inflamed and converted into a chronic abscess, which was opened externally. A fistulous opening resulted, which discharged a scanty colourless viscid fluid and gave exit to several small gall-stones. But after four months, with a fresh attack of biliary colic and vomiting, the concretion in the cystic duct was dislodged and projected into the common bile-duct, which it obstructed, as was proved by the motions becoming white and by bile being poured in large quantity through the fistulous opening. This state of matters continued for forty-one days (the patient passing white stools, but having scarcely a trace of jaundice), when there occurred another attack of biliary colic and vomiting, during which the concretion escaped into the duodenum and the flow of bile was restored to its proper channel. In this case, the quantity of bile secreted in twenty-four hours was fully two pints, although the patient did not weigh more than about 130 lbs. and was eating but a moderate diet. I have collected several other cases[1] of a similar nature, in which the

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[1] On this subject, see Bidder and Schmidt, Die Verdauungssäfte und der Stoffwechsel, 1852, p. 186; Dr. G. Scott, Beale's Archives of Medicine, 1858, vol i. p. 218; Carpenter's Human Physiology, 7th edit. p. 144.

[2] The weight of the liver of the dog is, from six observations of Bidder and Schmidt, as 1 to 26 of the whole body. According to Quain, the weight of the liver in man is as 1 to 36 of the whole body.

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[1] Haller, in his Physiology, alludes to the case of a man with a biliary fistula, from which 4 oz. of bile escaped in six hours ; but there is no mention as to whether or not the common bile-duct was closed. (Physiologia ; Berne, 1764, tome vi. p. 605.)

Heberden (Commentaries, 4th edit. p. 210) relates the case of a woman aged 50, who discharged 'a great quantity of yellow fluid for the space of four years,' from a fistula in the abdominal wall.

In the fourth volume of the Transactions of the College of Physicians Dr. Saunders recorded the case of a female aged 66, who discharged a large gall-stone through the abdominal parietes. For three weeks afterwards 'a very profuse discharge of bile ran perpetually from the wound,' although the common bile-duct remained pervious.

In the twenty-seventh volume of the Medico-Chirurgical Transactions, Mr. W. R. Barlow records the case of a man aged 54, in whom, in consequence of a strain, the common bile-duct became temporarily obstructed, probably by a gall-stone. The result was, that in twelve days thirteen pints of fluid accumulated in the gall-bladder. Dr. Owen Rees found, on analysis, that four-fifths of this fluid was pure bile, so that nearly one pint of bile must have been secreted in the day, although the patient was under the antiphlogistic treatment of the day (1844), and had been repeatedly bled from the arm and leeched. Moreover, as there was no external fistula, a large part of the secreted bile must have been absorbed.

In the thirty-fifth volume of the Medico-Chirurgical Transactions the case is recorded of a female aged 64, in whom the daily discharge of bile from a fistula was only 8 oz. The fistula was the result of a large gall-stone obstructing the common bile-duct and existed for six months, at the end of which time the woman died from exhaustion. The small quantity of bile in this case was accounted for by the circumstance of the patient being very poor and having insufficient food.

Dr. J. Hertz of Königsberg has recorded the case of a female aged 28, in whom the daily flow of bile from a biliary fistula was 18 fluid oz. It is doubtful, however, if all the bile secreted was really discharged from the fistula ; for, though the stools were of a pipe-clay colour, the fact that the flow of bile into the bowel results more or less corresponded with that observed in my patient; but I will not occupy time in reading the details. I will merely mention that Fauconneau-Dufresne, in his exhaustive memoir upon Biliary Calculi in the human subject, observes that from external biliary fistulæ, to which they now and then give rise, enormous quantities of bile may be discharged, so as to inundate the patient. He mentions one case in which the daily amount was two pints.[1]

Moreover, if, as seems probable, a large quantity of the bile which escapes into the bowel is reabsorbed, to be again passed through the liver, it is obvious that the quantity which escapes from a biliary fistula after closure of the common duct, in a dog or in man, is far short of what is secreted under ordinary circumstances. This inference is confirmed by the experiments of Schiff, who found that when a cannula was introduced into the gall-bladder, after closure of the common duct, the flow of bile was never so great as it was immediately after the operation, but that it was at once increased when bile was introduced into the veins or stomach.[2]

Although the amount of bile secreted daily must vary in different persons, and in the same person under different circumstances, being modified by the quantity and quality of the food, the activity of respiration, and other conditions, it is clear, from the facts now mentioned that but a small proportion of what is ordinarily secreted is discharged from the bowel. Berzelius found in 1,000 parts of fresh human fæces only 9 parts of a substance similar to bile, which, on the calculation that the daily fæces of a man weigh 5½ oz., would make a total of 24 gr. of dried bile in the day.[3] Now, assuming that the liver secretes only 40 oz. of bile in the day, containing only 5 per cent. of solid matter, which is considerably below the average proportion, the amount of dried bile secreted in one day would be 960 gr., or forty times the quantity discharged from the bowel. According to Bischoff, man discharges about 46 gr.

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was restored after closure of the external opening by needles (but not for six days, during which no jaundice appeared) seems to show that the obstruction of the common bile duct was not complete. (Berliner Klin. Wochenschrift, April 7, 1873.)

Lastly, Dr. Joseph Krumptmann has observed the case of a man who at 64 got a biliary fistula from gall-stones, which for ten years discharged bile up to half a pint daily. When the man died at 74, there were no signs of impaired nutrition, so that the reporter is probably justified in saying that only about one-fifth of the total quantity of bile secreted escaped by the fistula (London Med. Rec. April 30, 1873).

[1] Mém. de l'Acad. Royale de Méd.. 1847, tome xiii.

[2] Pflügers Archiv, 1870, p. 598.

[3] Budd. op. cit. p. 52.

of the (altered) biliary acids by the fæces per diem: whilst Voit's estimates give 170 gr. as the quantity daily formed by the liver; 124 gr. therefore must be otherwise disposed of.[1] Bidder and Schmidt have also found that not more than one-eighth of the sulphur of the bile is normally excreted with the fæces.[2] The bile-pigment is generally believed to be all voided by the fæces; but this is clearly not the case if there be any truth in the view already referred to, that urinary pigment is formed from bile-pigment; while the fact, familiar to all clinical observers, that the bile-pigment discharged from the bowel is greatly increased by calomel and other aperients, without any corresponding increase of secretion by the liver, also seems to show that, under ordinary circumstances, much of the bile-pigment secreted by the liver is not discharged with the fæces. It may be added that in carnivorous animals and in snakes, although bile-pigment is secreted in abundance by the liver, the quantity discharged with the fæces is even relatively less than in man.[3]

The question then arises as to what becomes of the bile which is not discharged from the bowel; and it is obviously one having an important bearing on the pathology of many cases of jaundice, as well as upon that of many functional derangements of the liver. The reply is to be found in the fact, that a large proportion of the bile secreted by the liver is again absorbed, either by the biliary passages, or by the mucous membrane of the bowel. From what is now known of the diffusibility of fluids through animal membranes, it is impossible to conceive bile long in contact with the lining membrane of the gall-bladder, bile-ducts, and intestine, without a large portion of it passing into the circulating blood. The constant secretion and reabsorption of bile is, in fact, merely part of that osmotic circulation constantly taking place between the fluid contents of the bowel and the blood, the existence of which is too little heeded in our pathological speculations[1] and in therapeutics, although attention was called to it nineteen years ago by Dr. Parkes, in the Gulstonian Lectures on Pyrexia delivered before this College. 'It is now known,' says Dr. Parkes,[2] 'that, in varying degrees, there is a constant transit of fluid from the blood into the alimentary canal, and as rapid reabsorption. The amount thus poured out and absorbed in twenty-four hours is almost incredible, and of itself constitutes a secondary or intermediate circulation never dreamt of by Harvey. The amount of gastric juice alone passing into the stomach in a day, and then reabsorbed, amounted in the case lately examined by Grünewald[3] to nearly twenty-three imperial pints. If we put it at twelve pints, we shall certainly be within the mark. The pancreas, according to Krœger, furnishes twelve pints and a half in twenty-four hours, while the salivary glands pour out at least three pints in the same time. The amount of the bile is probably over two pints. The amount given out by the intestinal mucous membrane cannot be guessed at, but must be enormous. Altogether the amount of fluid effused into the alimentary canal in twenty-four hours amounts to much more than the whole amount of blood in the body; in other words, every portion of the blood may, and possibly does, pass several times into the alimentary canal in twenty-four hours. The effect of this continual outpouring is supposed to be to aid metamorphosis; the same substance, more or less changed, seems to be thrown out and reabsorbed, until it be adapted for the repair of tissue or become effete.' How many times this cycle of movement is repeated, before the bile is extruded from the system, we have no means of knowing; but in the course of this osmotic circulation, much of the bile appears to become transformed into products which are eliminated by the lungs and kidneys, while at the same time this circulation assists in the assimilation of the nutritive materials derived from the food.[4]

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[1] Carpenter's Human Physiology, 7th edit. p. 435.

[2] Die Verdauungssäfte und der Stoffwechsel, 1852, p. 218.

[3] Liebig states that in the carnivora the whole of the bile is reabsorbed (Budd, op. cit. p. 51). Todd and Bowman also state that in carnivorous animals 'little or no bile is found in the excrements;' and 'in the boa, although the liver is large and no doubt secretes bile freely, the excrements contain no trace of bile' (Physiology, vol. ii. p. 259). Although the excrement of snakes, after feeding, does present a brownish colour and differs from the white masses of uric acid voided at other times, Marcet, who is an authority on the subject, states that the excrement of the boa is 'nearly entirely composed of urates.' (Philosophical Transactions, 1854, p. 279.)

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[1] The purging of cholera may result from a stoppage of this intestinal circulation—a diminished power of absorption rather than an increased exhalation from the bowel. Many facts prove that in cholera the power of absorption by the bowel is greatly impaired or abolished.

[2] Medical Times and Gazette, 1855, vol. i. p. 333.

[3] An account of this case, abstracted by me from Grünewald's Latin memoir, will be found in Beale's Archives of Medicine, vol. i. p. 270.—C. M.

[4] It may be thought improbable that the liver should secrete from the blood of the portal vein materials which are afterwards to be absorbed by the branches of the same vessel. But it has, perhaps, been too readily assumed, from the comparatively large size of the vena portæ, that it furnishes the materials for bile. Although, when

In the first place, it assists in the absorption of fat. It is a well-known clinical fact that, when the common bile-duct becomes obstructed from any cause in man, the fat throughout the body wastes. Many years ago, also, it was shown by Drs. Bright and Owen Rees that, in cases of this sort, an unusual quantity of fat may often be detected in the stools.[1] Bidder and Schmidt likewise found that, after applying a ligature to the gall-duct of a dog, the animal absorbed less fat than before, and there was also a diminution of fatty matter in the chyle in the thoracic duct; the amount absorbed was calculated from a comparison of the fat eaten with the amount passed in the fæces.[2] There are also grounds for thinking that the entrance of bile into the bowel facilitates the absorption of the albuminous constituents of the food. The bile neutralises the acid that passes from the stomach into the duodenum, and in doing so

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one vessel is diseased, its function may, in part, be performed by the other, it is probable that under ordinary circumstances the portal vein ministers chiefly to the assimilating functions of the liver, transporting to it the nutriment absorbed by its branches from the stomach and bowels; while the hepatic artery ministers to its secreting function, the biliary acids and bile-pigment being secreted from arterial blood, like urea and uric acid, which in the kidneys are secreted from the blood of the renal artery. This view is based on such facts as the following:—

1. In the Philosophical Transactions for 1793 a case is recorded where the portal vein passed direct to the vena cava inferior without entering the liver, and yet bile was found in the gall-bladder and intestines. Similar cases are referred to by Dr. Carpenter. (Principles of Human Physiology, 5th edit. p. 372.)

2. Many cases are on record where there has been complete obstruction of the portal vein from disease for some time before death, and yet bile has continued to be secreted. In 1856 Dr. Gintrac of Bordeaux collected thirty-four cases of obliteration of the portal vein in man, in not one of which was the biliary secretion interrupted. (L'oblitération de la veine porte, Bordeaux; see also Frerichs, op. cit. vol. i. p. 274; and Dickinson, Pathological Transactions, vol. xiv. p. 63.)

3. In animals the portal vein has been tied by Oré and other experimenters, and yet bile has been secreted. The reduction in the quantity may have been due to the febrile disturbance created by the operation, or to the absence of the fatty ingredients of the bile which may be contributed by the portal vein. (Oré, Journ. d'Anat. et Phys., 1864, p. 556; Carpenter's Human Physiology, 7th edit. p. 433; and Comparative Physiology, 4th edit. p. 424.)

4. Conversely, Kottmeier and Küthe found that no bile was secreted after ligature of the hepatic artery, although it is right to add that Schiff was unable to detect any diminution in a large dog, upon which he had performed the same operation; and Röhrig observed only a slight diminution in the flow of bile after obstruction of the hepatic artery. (Carpenter, Human Physiology, 7th edit. p. 433; and Röhrig, in Stricker's Jahrb. 1873, part ii.)

5. Portal blood contains very little cholesterin, but that of the hepatic artery contains a large quantity. (Trousseau, Clin. Lect. Syd. Soc. Ed. v. 140.)

[1] Guy's Hospital Reports, series i. vol. i. p. 610.

[2] Sanderson, op. cit. p. 505.

causes a precipitate of peptone. It is difficult to say what purpose is served by this precipitation; but some experiments of Bernard led him to the conclusion that gastric juice, when mixed with pancreatic juice and bile, has a more solvent action on albuminous substances than the gastric juice alone.[1] The passage of bile into the bowel appears also to be in some way essential to the formation of glycogen by the liver. In a number of experiments recently made upon cats, Dr. Wickham Legg found that the formation of glycogen was always arrested soon after ligature of the bile-duct; in one cat the diabetic puncture of the brain was made on the sixth day after ligature of the bile-ducts, but no sugar appeared in the urine.[2] The production of urea, however, in the liver appears to be quite independent of the passage of bile into the bowel, for in cases of permanent obstruction of the gall-duct the amount of urea voided in the urine may be quite normal.

But, lastly, there can be no doubt that bile is in part excrementitious, a portion of it being discharged from the bowel, and serving to rid the system of some of the products of waste of the blood and tissues. The biliary acid salts are decomposed, and, by their decomposition, they furnish the free alkali necessary for the precipitation of peptones and the saponification of the fatty matters; they are believed also to become further split up, the taurin, glycocin, and most of the cholic acid returning to the circulation, while a portion of the cholic acid is discharged with the fæces. The cholesterin is also decomposed in the bowels, and the products of its decomposition are discharged with the fæces. According to Dr. Austin Flint, jun., the cholesterin is converted into a substance which he has discovered in the fæces and designated stercorin; it appears also to be in some way related to another ingredient of the fæces discovered by Marcet and called excretin, although this contains sulphur. Marcet, at all events, made the observation, that in very young children cholesterin may take the place of excretin in the stools.[3] The bile-pigment also becomes changed in its passage through the bowel, the bilirubin becoming converted into biliverdin. Lastly, the bile, in its passage through the bowel, stimulates the peristaltic action of the gut; and, in virtue of its antiseptic property, which can be demonstrated out of the body, it prevents putrefactive fermentation of the intestinal contents and the excessive generation of gas.

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[1] Budd, op. cit., p. 50.

[2] On the Changes in the Liver which follow Ligature of the Bile-ducts. (St. Bartholomew's Hospital Reports, vol. ix. 1873.)

[3] Journal of the Chemical Society, October and November 1862.

From what has been stated, it follows that the functions of the liver may be summed up under three heads, viz.:—

1. The formation of glycogen, which contributes to the maintenance of animal heat and to the nutrition of the blood and tissues; and the development of white blood-corpuscles.

2. The destructive metamorphosis of albuminoid matter, and the formation of urea and other nitrogenous products, which are subsequently eliminated by the kidneys, these chemical changes also contributing to the development of animal heat.

3. The secretion of bile, the greater part of which is reabsorbed, assisting in the assimilation of fat and peptones and probably in those chemical changes which go on in the liver and portal circulation: while part is excrementitious, and, in passing along the bowel, stimulates peristalsis and arrests decomposition.

B. FUNCTIONAL DERANGEMENTS OF THE LIVER.

Keeping in view the functions of the liver in health, which I have endeavoured thus briefly to sketch, we shall be the better able to discuss the symptoms resulting from derangements of these functions. The few medical writers who have described the functional derangements of the liver have, like the late Dr. Copland,[1] arranged them under the three following heads: 1. Diminished secretion of bile; 2. Increased secretion of bile; 3. Secretion of morbid or altered bile. But this classification fails to recognise the most important functions of the liver; and, from what has been stated, it follows that the quantity and quality of the bile discharged from the bowel, upon which the classification is based, are no certain tests of the amount and quality of the bile secreted by the liver. The quantity secreted being the same, the quantity discharged from the bowel will vary with whatever stimulates or impedes absorption. Any substance like calomel, or podophyllin, or certain articles of diet, which irritates the commencement of the small intestine, will sweep along the bile before there is time for its absorption, and thus cause an increased flow from the bowel, without the secretion by the liver being necessarily increased. Moreover, it must often be impossible to say whether the morbid or altered appearances of the bile in the fæces be due to a vitiated bile, or to changes which the bile has undergone in its passage through the bowel. For these reasons, I have ventured to suggest another classification of the functional derangements of the liver, based upon what are now believed to be the normal functions of the gland, and upon the symptoms which a disordered liver may excite in the different physiological systems of the body.

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[1] Medical Dictionary, vol. ii. p. 723.

Classification of the Functional Derangements of the Liver.

I. Abnormal Nutrition.
II. Abnormal Elimination.
III. Abnormal Disintegration.
IV. Derangements of the Organs of Digestion.
V. Derangements of the Nervous System.
VI. Derangements of the Organs of Circulation.
VII. Derangements of the Organs of Respiration.
VIII. Derangements of the Genito-Urinary Organs.
IX. Abnormal Conditions of the Skin.

I. *Abnormal Nutrition.*

Functional derangement of the liver may lead directly to (1) an abnormal deposition of fat, or to (2) the opposite condition of emaciation. Indirectly, also, the nutrition of the body may become seriously impaired from derangements of the disintegrative functions of the liver.

1. *Corpulence*, by which so many persons are inconvenienced, owes its origin to different causes. We know that it is very liable to occur in persons who eat much fat and take little exercise. In this case, the lacteals absorb more fat from the bowel than is sufficient to supply the carbon consumed in respiration, or there is a deficient consumption of fat in the system. Many facts, however, show clearly that the accumulation of fat is not due solely to an increased supply of fatty matter in the food and its absorption by the lacteals. Thus, of two persons consuming the same amount of fat and taking the same amount of exercise, one will accumulate fat and the other will not; and in many persons the accumulation of fat appears to be one of the characteristics of old age, just as other persons in growing

old wither and dry up. Again, some persons, with every care as to diet, cannot avoid getting fat; while others, consuming much fat, as well as saccharine and starchy matter, remain permanently thin. And, thirdly, it is a fact established by many observations both in man and in the lower animals, that fat is formed in large quantity from a diet containing much starch and sugar, and, from what has already been stated, it seems probable that the liver is mainly concerned in this transformation. Some of the fattest persons I have met with have been females, who have for a long time eaten little or no fat or oily matter, and who indeed have taken very little solid food, but who have contracted the habit of drinking frequently some mixture of alcohol and sugar, such as we find in beer, champagne, and other wines, and who at the same time have taken little exercise and have suffered from symptoms of deranged liver. M. Dancel relates the case of a young lady, who, finding herself getting stout, and with the object of preserving her symmetry, fasted, as she thought, four days in the week upon champagne and *marrons glacés*; but with this diet she accumulated fat with frightful rapidity, and it was only after returning to a more rational diet that she regained her normal figure.[1] The tendency to accumulate fat, or the reverse, appears to be due to some constitutional peculiarity transmissible by parents to their offspring; and, from what we know of the functions of the liver, it is highly probable that this is the organ mainly at fault. Possibly, there may be an abnormal proneness, or the reverse, to the conversion of glycogen into fat, in the manner suggested by Dr. Pavy; or, from some derangement of the liver, a larger proportion of the glucose derived from the food may be directly converted into fat than in health; or, in some instances, obesity may be due to a deficient oxydation of fatty matter. Although we can only as yet speculate as to the precise nature of the morbid process, we know that in animals eating much farinaceous, saccharine, or oleaginous food, the proportion of fatty particles in the secreting cells of the liver is much greater than in animals moderately fed and taking much exercise.

Persons of a corpulent tendency are very liable to flatulence, constipation, heaviness and weariness after meals, and other symptoms of hepatic derangement. After a time, they lose all appetite for solid food; they complain of great prostration and of sensations of sinking, which prevent their making much muscular exertion and often encourage them in habits of tippling.

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[1] Quoted by Dr. T. K. Chambers (Clinical Lectures, 1864, p. 547). I have failed to find the case in the Traité Théorique et Pratique de l'Obésité, par F. Dancel; Paris, 1863.

Lastly, by improving the condition of the liver not only these symptoms, but the corpulence, will often disappear.

2. *Emaciation* may be induced by functional derangement of the liver in different ways.

a. In consequence of a deficient formation of bile, or of its impeded passage into the bowel, the assimilation of fatty and albuminous matters is interfered with. It is true that cases are occasionally met with in which the common gall-duct has been completely and permanently closed by a gall-stone, so that no bile could flow into the intestine, and yet the body has been tolerably well nourished even after two or three years.[1] There are, however, few exceptions to the rule, that permanent closure of the common bile-duct destroys life in the end—usually in little more than twelve months, if not before—by causing a gradual impairment of nutrition. Most patients with obstruction of the bile-duct dislike fat and cannot assimilate it, the fatty matters of the ingesta being, as already stated, discharged with the fæces. There are also reasons for thinking that the absence of bile from the bowel interferes with the absorption of peptones; while Dr. Legg's observation that ligature of the hepatic duct in animals arrests the glycogenic function of the liver makes it probable that, when the flow of bile into the bowel is impeded, the nutrition of the body suffers in other ways than by the deficient absorption of fat and albumen. Obstruction of the bile-ducts also leads to a deficient formation of blood-corpuscles and anæmia. A deficient secretion or a morbid quality of bile may possibly lead to similar results, though in a less degree.

b. But, secondly, emaciation may result from derangement of the glycogenic function of the liver. *Diabetes*, in fact, may be said to be in most instances a derangement of the glycogenic function of the liver. It would be out of place here to consider in detail what are now believed to be the various causes of glycosuria;[2] but, briefly, they may be said to come under one of the three following heads.

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[1] Budd, op. cit. p. 49.

[2] These are well given by Dr. T. L. Brunton in his Lectures in the British Medical Journal for January and February 1874.

1. *Imperfect glycogenesis* in the liver. One function of the liver in health appears to be to prevent the immediate passage into the blood of the glucose derived from the food, by converting it into glycogen. Thus, Bernard has shown that, if a ligature be applied to the portal vein in an animal, so that the intestinal blood, rich in glucose, reaches the systemic circulation without passing through the liver, sugar at once appears in large quantity in the urine; and in man diabetes has been known to occur when the portal vein has been obliterated.[1] Again, if sugar be injected into the crural vein or into the rectum, it appears in the urine; but, if it be injected slowly into the portal vein, the urine will contain none.[2] The capability of the liver to convert sugar into glycogen is not unlimited. When, therefore, sugar is swallowed in excess of a certain quantity, it appears in the urine; and the same thing happens when much sugar or starch is taken after long fasting, owing to the rapidity of intestinal absorption; or when, in consequence of disease, old age, injury of the liver, unsuitable food, or some other cause, the glycogenic function of the liver is impaired. Glycosuria, often temporary, from some of these causes is not uncommon. According to Bence Jones, in half of the cases of diabetes, the disease consists in an arrest of change in the food-sugar.

2. *An increased conversion of glycogen into sugar*, the destruction of the sugar remaining unaltered. This appears to be the main cause of persistent glycosuria, or diabetes. In considering the functions of the liver, it was pointed out that the glycogen formed in it was partly converted into sugar, which disappeared in the lungs and muscles, but was probably mainly destined to support the nutrition of the blood and tissues throughout the body. Whatever, then, favours the flow of sugar from the liver into the blood, to an extent greater than can be consumed in the lungs and muscles, will lead to an excess of sugar in the blood and its appearance in the urine, and will to a corresponding extent interfere with the nutrition of the body. Now, whatever quickens the circulation of blood through the liver, particularly in the hepatic arteries, favours the conversion of glycogen into sugar, possibly by increasing the amount of

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[1] See abstract of Lectures in London Medical Record for October and November 1873.

[2] Pavy, Nature and Treatment of Diabetes, 2nd edit. 1869, pp. 142–43; and Bence Jones, Lectures on Pathology and Therapeutics, 1867, p. 42.

albuminoid ferment already referred to; and accordingly, whatever paralyses the vaso-motor nerves of the hepatic vessels, either directly or indirectly, dilates these vessels, produces an increased flow of blood through them, and so leads to diabetes. The tendency of glycogen to become converted into sugar appears to be moderated in health by some nervous influence, on the removal of which the blood becomes surcharged with sugar which enters the urine. The recent observations of Dr. Pavy[1] show also that oxygenated blood in some manner influences the liver so as to lead to the production of glycosuria, which is accordingly induced when blood reaches the portal system without having become dearterialised in the natural way, owing to vaso-motor paralysis of the vessels of the chylo-poietic viscera. Thus it is that diabetes is produced by irritation of the roots of the pneumogastric nerve in Bernard's 'diabetic puncture,' by certain injuries and diseases of the brain and spinal cord in man, by distress and anxiety of mind, by poisoning with woorari, and by injuries or lesions of the peripheral extremities of the pneumogastric nerve in the lungs, liver, stomach, or intestines.

3. *Diminished destruction of sugar.* If the sugar into which the glycogen formed in the liver is converted did not disappear from the blood, it would necessarily form one of the normal constituents of the urine. Hence it cannot be denied that the pathological presence of sugar in the urine may possibly depend in some cases upon a failure of the conditions under which the normal transformation of the sugar takes place. We are, however, in great measure ignorant as to what these conditions are. A number of experiments make it probable that the sugar in the blood, under the influence of a ferment, is converted into lactic acid and glycerin, which undergo combustion and so maintain the animal heat; and it is believed that, when this ferment is absent, the sugar is not decomposed, but is excreted in the urine. Whether this be the true explanation or not, many facts seem to show that glycosuria may result from a morbid state of the blood. Thus the introduction into the blood of ammonia, ether, chloroform, or phosphoric acid is followed by glycosuria; while the introduction of carbonate of soda will prevent it. Glycosuria also has been repeatedly observed in cases of pneumonia, whooping-cough, and phthisis, which lead to deficient oxygenation of the blood. It is worth

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[1] Proc. Roy. Soc. Nos, 163, 164. 1875.

adding that there has long been supposed to be an antagonism between diabetes and gout. Gouty dyspepsia and actual gout have been observed to cease on the supervention of diabetes; and it was pointed out by Sir C. Scudamore that the Scotch were much more liable to diabetes, and less prone to gout, than the inhabitants of England.

c. It is not improbable that other wasting diseases are in their origin connected with some functional disorder of the liver. When there is derangement of the disintegrative function of the liver, the blood and fluid effused from it become loaded with effete matter, as the result of which the nutrition of the tissues is often impaired and the body wastes. In *phthisis*, again, long before tubercle is deposited in the lungs, there is evidence of deficient assimilation of nutriment and imperfect sanguification—functions in which we know that the liver is deeply concerned. Again, the protracted purulent discharge which usually precedes *waxy disease* may entail a hasty and imperfect sanguification, resulting in anæmia and the formation of an albuminous material little capable of organisation.

II. *Abnormal Elimination.*

In discussing the functions of the liver, I stated that bile is, in part, excrementitious, although this is probably far from being its chief use in the economy of digestion. In accordance with the belief still held by many members of the profession engaged in practice, that the chief function of the liver is to excrete bile, the retention of bile in the blood and tissues is believed to give rise to serious symptoms. For example, when such symptoms as delirium, stupor, muscular tremors, subsultus, carphology, paralysis of the sphincters, coma, convulsions, a dry brown tongue, and other phenomena of the 'typhoid state' supervene in any case of jaundice, in acute atrophy of the liver, or in the advanced stage of cirrhosis, it is customary to attribute them to poisoning of the blood with retained bile. Experiments, also, have been performed on animals, with the object of showing that bile is a deadly poison. But that dogs should die after injection into the cellular tissue of the bile of other dogs admits of another explanation than that of the essential elements of bile being a poison. The injection of decomposing mucus would probably produce a like result. Pure bile, from which all mucus has been removed, has been repeatedly injected into the large veins of dogs by Frerichs and other observers, without any cerebral symptoms or bad results ensuing, except that in some instances death has been caused by the entrance of air into the veins.[1] The operation has even been repeatedly performed on the same animal without any lasting injury; but it is scarcely necessary to have recourse to experimental researches on the lower animals for evidence on the matter, and in all these experiments there are sources of fallacy. Every medical practitioner must be familiar with the fact, that the blood and tissues of man may be saturated with bile for months, without cerebral or any other symptoms of blood-poisoning resulting, so long as the glandular tissue of the liver is not destroyed and the kidneys continue to perform their functions. Bearing such cases in mind, it is difficult to believe that bile is a deadly poison.

Reference has already been made to the views of an American physiologist, Dr. Austin Flint, jun., who has written a work to show that the cerebral symptoms which occasionally occur in jaundice and in structural diseases of the liver are due to the retention in the blood of cholesterin, or to what he has designated '*Cholestearæmia*.'[2] Dr. Flint regards cholesterin as an excrementitious product of nervous tissue, the elimination of which from the body he believes to be one of the functions of the liver. Having arrived in the bowel, the cholesterin, according to him, is converted into stercorin, and therefore it is not found in the fæces; but when retained in the blood and tissues, he believes it to be a virulent poison like urea. But if the non-excretion of all the elements of bile do not give rise to cerebral symptoms, it is difficult to understand how these symptoms can result from the retention of cholesterin alone. In cases, for instance, of permanent closure of the bile-duct, cholesterin is not discharged from the liver into the bowel; it does not accumulate in the biliary passages; nor, if it be present in the blood, does it necessarily give rise to cerebral symptoms.

From what has been stated, I think it must be clear that the cerebral symptoms which occasionally supervene in certain morbid states of the liver are independent of the non-excretion of bile, or of any of its component parts. The real cause of these cerebral symptoms will be referred to hereafter.

The symptoms usually associated with a deficient excretion of bile are an irregular, usually costive, state of the bowels, the stools being insufficiently coloured with bile and of a pale yellow, drab, or whitish colour; loss of appetite; a white or yellowish furred tongue; a disagreeable, often bitter, taste in the mouth, especially in the morning; flatulence; a sallow or muddy tint of skin (indicating, unless there be concurrent hyperæmia of the liver, anæmia rather than jaundice); dingy conjunctivæ; languor and disinclination for exertion; frontal headache; dulness and heaviness, drowsiness after meals, great depression of spirits, and sometimes hypochondriasis; and frequent deposits of lithates in the urine on cooling. These symptoms are very apt to be induced, especially towards middle life, by sedentary or indolent habits, the habitual use of rich or indigestible food, neglect of the bowels, great or protracted anxiety of mind, or by a general want of vigour, consequent upon disease of the heart or of some other organ; and the tendency to them is in many cases inherited. They are commonly, and perhaps correctly, ascribed to what is called 'torpor of the liver;' but the non-excretion of bile may possibly be merely one of the symptoms, rather than the cause, of the morbid state, the real cause being the retention in the system, not of bile, but of those products of disintegration which it is the purpose of the kidneys to eliminate. At the same time, it is very probable that engorgement of the liver with bile interferes with the normal processes of disintegration of albumen which take place in the gland.

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[1] Diseases of Liver, Sydenham Society's translation, vol. i. p. 395.

[2] Op. cit.

LECTURE XV.

THE CROONIAN LECTURES ON FUNCTIONAL DERANGEMENTS OF THE LIVER.

III. ABNORMAL DISINTEGRATION. 1. LITHÆMIA; 2. GOUT; 3. URINARY CALCULI; 4. BILIARY CALCULI; 5. DEGENERATIONS OF THE KIDNEYS AND ALBUMINURIA; 6. STRUCTURAL DISEASES OF THE LIVER; 7. DEGENERATIONS OF TISSUE THROUGHOUT BODY; 8. LOCAL INFLAMMATIONS; 9. 'CONSTITUTIONAL DISEASES.'—IV. DERANGEMENTS OF ORGANS OF DIGESTION. 1. TONGUE; 2. APPETITE; 3. TASTE; 4. DYSPEPSIA; 5. CONSTIPATION AND DIARRHŒA; 6. VITIATED STOOLS; 7. INTESTINAL HÆMORRHAGE; 8. HÆMORRHOIDS; 9. HEPATIC PAIN; 10. JAUNDICE, ITS PATHOLOGY.—V. DERANGEMENTS OF THE NERVOUS SYSTEM. 1. ACHING PAINS IN LIMBS; 2. BURNING PATCHES: 3. NEURALGIA; 4. CRAMPS; 5. HEADACHE—MEGRIM; 6. VERTIGO; 7. CONVULSIONS; 8. MANIA; 9. PARALYSIS: 10. NOISES IN THE EARS; 11. SLEEPLESSNESS; 12. DEPRESSION OF SPIRITS; 13. IRRITABILITY; 14. THE TYPHOID STATE.

MR. PRESIDENT, FELLOWS OF THE COLLEGE, AND GENTLEMEN,—In my first lecture I endeavoured to show that the secretion of bile was, perhaps, the least important of the functions of the liver; but that, in the first place, this organ contributed greatly to the processes of sanguification and nutrition of the tissues; and, secondly, that it was probably the chief seat of the disintegration of albuminous matter; while even the secretion of bile, although in part excrementitious, was mainly destined to assist the assimilation of the food. If these views were correct, I pointed out that the existing classification of the functional derangements of the liver, founded on the quantity or the quality of the bile in the stools, had become obsolete; and I proposed to substitute another, founded on the more important functions of the liver and on the derangements which the liver excites in other organs of the body. I then described some of the derangements of nutrition and elimination traceable to the liver, and I now proceed to consider the more important disorders coming under the head of Abnormal Disintegration.

III. *Abnormal Disintegration.*

Modern investigations, pathological as well as physiological, go far to prove that one of the chief functional derangements of the liver, if it be not the foremost of all, is an imperfect disintegration of albuminous matter, or its non-conversion into a soluble product (urea), which can be readily excreted by the kidneys. A strong corroboration of the correctness of this view is furnished by the disease known as acute atrophy of the liver, to which I have already had occasion to refer. In this disease there is a rapid destruction of the secreting tissue of the liver, and the result is a disappearance of urea from the urine, and the substitution for it of leucin ($C^6H^{13}NO^2$) and tyrosin ($C^9H^{11}NO^3$), products of the disintegration of albumen more complex and less oxydised than lithic acid ($C^5H^4N^4O^3$) and urea (CH^4N^2O), which are also found in abundance in the wasted tissue of the liver (figs. 35, 36, and 37). The substitution of leucin and tyrosin for a portion of the urea of the urine takes place in other diseases of the liver, in which the destruction of the hepatic tissue is less rapid and less extensive than in

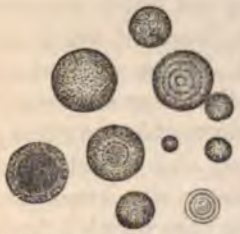

FIG. 35.—Laminated crystalline masses of Leucin.

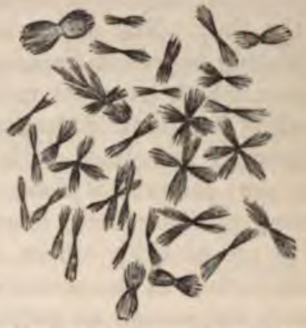

FIG. 36.—Needle-shaped crystals of Tyrosin, adhering in bundles and in stellate groups.

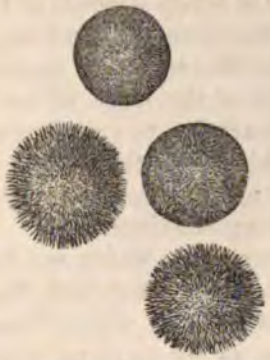

FIG. 37.—Globular masses composed of acicular crystals of Tyrosin.

acute atrophy, as, for example, in certain cases of cirrhosis and of obstruction of the common bile-duct. I have known it also to occur in certain febrile diseases, such as typhus and enteric fever, in which the hepatic tissue appears, in consequence of the increased work thrown upon it, to undergo partial disintegration.[1] But these important changes in the urine, so far as we know, only occur in cases where there is structural change of the liver. The urine, however, is liable to other changes indicating imperfect disintegration or oxydation of albuminous matter, which are much more common, and which are also the result of functional derangement of the liver, but which are not necessarily associated with structural disease of that organ. The most common of these changes in the urine are deposits on cooling of lithic acid, lithates, and pigmentary matters; but there are probably others, less frequent and as yet but little studied, such as the presence of xanthin, cystin, kreatinin, etc. I need not remind an audience such as that which I have the honour to address, that deposits in the urine of lithic acid or lithates are not due to any morbid condition of the kidneys. What I wish to insist upon is, that the frequent occurrence of these deposits in the urine ought always to be regarded as a sign of functional derangement of the liver, arising from causes, sometimes temporary, at other times more or less permanent. Excluding those cases in which deposits of lithic acid or lithates are thrown down in the urine not until twelve or twenty hours after its emission, as the result of spontaneous changes, to which Scherer has given the name of acid urinary fermentation,[2] and those which are due to a marked deficiency of urinary water, deposits of lithic acid, lithates, and abnormal pigments, which appear in the urine as soon as it cools, are chiefly met with under the following conditions:—

1. In febrile diseases, in which we know that the liver becomes enlarged and congested, and its gland-cells loaded with minute granules, and in which there always is an increased disintegration of albuminous matter. Everyone, for example, is familiar with the copious deposits of lithates which are so common during an attack of ordinary febrile catarrh.

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[1] See Murchison on The Continued Fevers of Great Britain, 2nd edit. 1873, pp. 157, 210, 255, 533, 629.

[2] Ann. d. Chemie und Pharm., Bd. 42, s. 171. Dr. Bence Jones's experiments throw doubts upon the occurrence of the acid fermentation in the urine described by Scherer, Lect. on Path. and Therap. 1867, p. 216.

2. In many structural diseases of the liver, and particularly in those which are characterised by an increased amount of blood in the organ, such as inflammation, cirrhosis, cancer, and simple hyperæmia, whether mechanical or active.

3. In functional derangements of the liver, either temporary or persistent.

To this third cause of an excess of lithic acid in the urine I wish now to restrict my remarks. What I desire to maintain is, that lithuria, like glycosuria, is very often due to a functional disease of the liver, although even glycosuria is still ranged in some text-books with albuminuria and diseases of the kidneys. In other words, abnormal disintegration of albuminous matter in the liver may lead to a morbid condition of the blood and of the entire system, which often manifests itself in lithuria. This morbid state of the blood I propose to designate Lithæmia.[1]

1. *Lithæmia.*—When oxydation is imperfectly performed in the liver there is a production of insoluble lithic acid and lithates, instead of urea which is the soluble product resulting from the last stage of oxydation of nitrogenous matter. Persons who habitually enjoy the best of health are liable to deposits of lithates in the urine after a surfeit of food, or even after partaking moderately of one of the fashionable dinners of the age. When more food is taken into the blood than is necessary for the nutrition of the tissues, the excess is thrown off by the kidneys, lungs, and skin in the form of urea, carbonic acid and water, or in the imperfectly oxydised forms of lithic acid and oxalic acid. Under these circumstances, an excess of work is thrown upon the liver and the other glandular organs, and one result is that a quantity of albumen, instead of being converted into urea, is discharged by the kidneys in the less oxydised form of lithic acid or its salts. But what in most persons is an occasional result of an extraordinary cause is in some almost a daily occurrence, either from the food being always excessive in amount or unduly stimulating, or from some innate defect of power, often hereditary, in the liver, in virtue of which its healthy functions are liable to be deranged by the most ordinary articles of diet. Most persons appear to

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[1] It has been pointed out to me that Dr. Austin Flint has proposed the term 'uricæmia' to designate the excess of uric acid in gout and in cases of lead-poisoning (Principles and Practice of Medicine, 3rd edit. Philadelphia, 1868, p. 86). The term which I have employed appears to me to be preferable.

have more liver, just as they have more lung, than is absolutely necessary for the due performance of its functions. But in others, not unfrequently the offspring of gouty parents, the organ in its natural condition seems only just capable of performing its healthy functions under the most favourable circumstances, and functional derangement is at once induced by articles of diet which most persons digest with facility. This functional derangement may manifest itself by various symptoms of 'indigestion,' by disturbances of circulation and of other physiological systems, but especially by deposits of lithic acid, lithates, and pigments in the urine. These deposits, it is true, are often absent, and yet the urine may contain a great excess of lithic acid. Indeed, as Dr. Bence Jones[1] has shown, clear urine sometimes contains more lithic acid than that which thickens on cooling. But the frequent deposit of lithates shows that oxydation is less perfect than it ought to be. Very often, however, these urinary deposits exist for years, without the patient experiencing any general or local discomfort. But sooner or later, often about middle life, lithic acid and lithates are formed in such excess that they cannot be eliminated by the ordinary channels, and they accumulate and create disturbance in different parts of the organism, and then the urinary deposits begin to attract more attention than they had previously done, from their being accompanied by symptoms of indigestion, or from the evidence of some serious local mischief, the onset of which had been insidious. These symptoms are all the more likely to occur if the patient be what is commonly known as 'a generous liver,' if he take little exercise in the open air, or if he have much mental work. Of the symptoms referred to, the most common are the following:—

a. A feeling of weight and fulness at the epigastrium and in the region of the liver.

b. Flatulent distension of the stomach and bowels.

c. Heartburn and acid eructations.

d. A feeling of oppression and often of weariness and aching pains in the limbs, or of insurmountable sleepiness after meals.

e. A furred tongue, which is often large and indented at the edges, and a clammy, bitter, or metallic taste in the mouth, especially in the morning.

f. Appetite often good; at other times anorexia and nausea.

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[1] Philosophical Transactions, 1849, part ii. p. 249.

g. An excessive secretion of viscid mucus in the fauces and at the back of the nose.

h. Constipation, the motions being scybalous, sometimes too dark, at others too light, or even clay-coloured. Occasionally attacks of diarrhœa alternating with constipation, especially if the patient be intemperate in the use of alcohol.

i. In some patients, attacks of palpitation of the heart, or irregularity or intermission of the pulse.

k. In many patients, occasional attacks of frontal headache.

l. In many patients, restlessness at night and bad dreams.

m. In some patients, attacks of vertigo or dimness of sight, often induced by particular articles of diet.

All these symptoms are liable to occasional aggravation from errors in diet. Gradually the patient is taught by experience to become more careful as to what he eats or drinks. One thing after another he is compelled to give up. First, he renounces malt liquors; then he discovers that Port wine, Madeira, Champagne, and Burgundy disagree, and he betakes himself for a time to dry Sherry: but at length this does not suit, and after an interval, during which a trial is made of Claret or Hock, the patient, probably under medical advice, finds temporary relief from the substitution for wines of brandy or whisky largely diluted with water. At last, unless he be misled by the fashionable, but to my mind erroneous, doctrine of the present day, that alcohol in one form or another is necessary for digestion or to enable a man to get through his mental or bodily work, he finds that he enjoys best health when he abstains altogether from wine and spirits and drinks plain water. The patient goes through a like experience with regard to solid food; one dish after another, very often what he likes best, has to be given up, until at length, if he be well advised and have the sense to follow the advice, he restricts himself to the plainest food in moderate quantity. As a rule, those articles of diet are most apt to disagree which contain much saccharine or oleaginous matter, and not, as might perhaps have been expected, nitrogenous food, plainly cooked. In most of these cases the digestion appears to be strongest in the morning, and the patient suffers from late dinners or suppers.

The picture which I have thus imperfectly drawn represents a morbid condition extremely common in this country, which is associated with a constant tendency to the deposit of lithic acid or lithates in the urine, and which, for the reasons already given, I believe to originate in functional derangement of the liver.

2. *Gout.* But, secondly, you cannot fail to recognise that this picture represents a train of symptoms very common in gouty people, and to which the terms 'gouty dyspepsia,' 'latent gout,' 'suppressed,' 'anomalous,' or 'irregular gout' have been applied; although what I desire to maintain is, that they are also very common in persons, who neither inherit articular gout nor ever have it themselves. Gout itself, however, is merely one of the results of lithæmia. Thanks to the researches of our colleague Dr. Garrod, we now know that gouty inflammation of a joint is due to various exciting causes, which it is needless here to enumerate, determining a local deposit of lithate of soda, which has previously existed in abnormal quantity in the blood, as well as in the fluid that exudes from it into all the textures of the body. The accumulation of this substance in the blood will, no doubt, be favoured by non-elimination consequent upon disease of the kidneys; but in most cases of gout the kidneys are in the first instance healthy, and the presence in the blood or tissues of lithic acid or its salts is the result of imperfect digestion, or more strictly of functional derangement of the liver. Articular gout is, so to speak, a local accident, which, though sometimes determined by an injury, yet may occur at any time in persons in whom the normal process by which albuminous matter becomes disintegrated in the liver into urea is persistently deranged. In other words, gout, like diabetes, is the result of a functional derangement of the liver; and just as we found that in many persons in whom there is no evidence of articular gout, an innate defective power of the liver, in virtue of which its functions are deranged with unusual facility, is often transmitted by parents to their offspring, so gout, which is one of the consequences of that condition, comes to be an hereditary disease. I hold, therefore, that what is called a 'gouty diathesis' always indicates, and is the result of, hepatic derangement, and that many symptoms commonly referred to gout would be more correctly ascribed to disorder of the liver.

3. *Urinary Calculi* are another consequence of lithæmia, and therefore of functional derangement of the liver. Of the concretions which form in the urinary passages the great majority consist, in the first instance at all events, of lithic

acid or its salts. According to Dr. William Roberts, lithic acid constitutes five-sixths of all renal concretions and of vesical calculi which have only recently descended from the kidney.[1] The circumstances favourable to the precipitation of lithic acid are catarrhal and other morbid states of the urinary passages and an acid condition of the urine, but mainly an excessive elimination by the kidneys of free lithic acid, which had previously existed (either free or combined) in excess in the blood, and which we have seen to be formed, mainly at all events, in the liver. There are also good reasons for believing that renal calculi composed of other substances than lithic acid have a hepatic origin. Cystin ($C^3H^7NSO^2$), for example, which represents a different process of oxydation from that which produces lithic acid, closely resembles taurin, and, like it, contains a large proportion of sulphur;[2] and, moreover, it has been found by Scherer in the livers of patients suffering from enteric fever.[3] It would seem, therefore, that those rare renal calculi which are composed of cystin are due in the first instance to some functional derangement of the liver. Xanthin ($C^5H^4N^4O^2$), again, of which a few renal calculi, chiefly in young people, are composed, also appears to arise from imperfect oxydation of the products of albuminous matter. It differs from lithic acid only in containing one atom less of oxygen; so that it also is probably formed in the liver, in which organ it has been found by Scherer, as well as in the blood, spleen, and muscles.[4] Even oxalate of lime, which Dr. Bence Jones found, either alone or combined with other substances, in 163 out of 450 urinary calculi,[5] may in some way be connected with functional derangement of the liver, although the evidence on this point is less clear than in the case of other urinary concretions. I do not forget that it was maintained by so eminent an authority as our colleague Dr. Owen Rees, in the Croonian Lectures delivered before this College in 1856, that oxalic acid is never excreted from the blood, but is always formed in the urine after its secretion by decomposition of lithic acid; and that this opinion has more recently been advocated by another eminent Fellow of this College, Dr. Basham.[6] Facts, however, usually after food, is loaded with albumen, whereas at others it contains none; and this state of matters may last for many months, and then completely disappear.[1] Now it is not contended that in all these cases the liver is the organ primarily at fault, but certainly in some there is good reason for believing it to be so, the albuminuria being unattended by any other symptom of renal disease, varying greatly in quantity and sometimes absent, and the urine being of normal quantity, of high specific gravity, and habitually loaded with lithates, lithic acid, oxalates, and pigments,[2] and there being very often cutaneous eruptions, dyspepsia, and other evidence of hepatic derangement. I have met with several instances of this sort where the patient was subject to severe attacks of what at first seemed to be hepatic colic, but where there was no jaundice and the paroxysm was followed by a temporary yet extraordinary increase of lithates and albumen in the urine. Lastly, so often have I observed albuminuria associated with hepatic disorder, which has disappeared completely and permanently when this has been set to rights, that I have little doubt that we have in the liver a cause of albuminuria to which attention has not hitherto been sufficiently directed.[3] The pathology of the albuminuria in these cases may be similar to that of certain cases of diabetes already referred to, the liver having too much work to do, and permitting some albumen to pass through in a form which cannot be assimilated; or possibly there may be some defect in the destructive functions of the liver, in consequence of which the albuminous matter, instead of being converted into urea, does not even reach the stage of lithic acid. It is possible that in many of the cases now referred to the albuminuria may indicate an early stage, not yet described, of the contracted or gouty kidney, yet it is certain that the symptom may persist or recur during many years without any but do not cure the disease; whereas more permanent results are obtained by the use of the waters of Friedrichshall, Carlsbad, and Püllna, which produce activity in all the digestive organs, and eliminate by other channels the waste matters previously thrown out as lithic acid by the kidneys, but which mainly act by relieving the overloaded liver and restoring its normal functions.

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[1] Urinary and Renal Diseases, 2nd edit. 1872, p. 270.

[2] Roberts, op. cit. 2nd edit. 1872, p. 84.

[3] Archiv. f. Path. Anat. Bd. x. p. 228.

[4] Roberts, op. cit. p. 88.

[5] Lectures on Pathology and Therapeutics, 1867, p. 99.

[6] Renal Diseases, 1870, p. 187.

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[1] See some interesting cases of this sort recorded by the late Dr. J. Warburton Begbie, Edin. Med. Journ. April 1874. In a letter to Dr. Begbie respecting one of these cases, Dr. George Johnson observes: 'I have often met with cases in which the urine has been albuminous only after food and exercise.' In these cases both Dr. Begbie and Dr. Johnson coincided in thinking that the albuminuria was allied to the albuminuria of indigestion, and did not indicate structural disease of the kidneys.

Dr. James Finlayson has found numerous hyaline tube-casts in non-albuminous urine which was loaded with urea and lithates. Brit. and For. Med. Chir. Rev. January 1876.

[3] Dr. W. Whitla has recently called attention to the influence of the liver in determining both anæmia and albuminuria. Dublin Journ. of Med. Science, February 1876.

4. *Biliary Calculi*, which consist for the most part of cholesterin and bile-pigment, are another result of functional derangement of the liver. They are chiefly met with in persons of middle or advanced life who have led sedentary lives, and they are particularly common in those who have lived too well and eaten much saccharine food, and who are the subjects of lithæmia. It may be difficult to explain how the functional derangement of the liver which results in lithæmia should also lead to the formation of concretions composed of cholesterin and bile-pigment in the biliary passages; but the frequent concurrence of the lithic acid dyscrasia with gall-stones is a clinical fact which I have had many opportunities of verifying. This observation accounts for the frequent occurrence of biliary calculi in gouty people, and it also explains the frequent coincidence in the same individual of gall-stones and urinary calculi. I cannot agree with Frerichs in regarding this coincidence as purely accidental.[1] Many years ago Baglivi and Morgagni insisted on the frequent coexistence of urinary and biliary calculi. The late Dr. Prout also remarked that the formation of gall-stones of cholesterin was frequently associated with a tendency to lithic acid deposits in the urine; and Dr. George Budd, in his classical work on Diseases of the Liver, states that, 'the habit of drinking porter, which frequently leads to lithic acid deposits and to the most inveterate forms of gout in persons who inherit no disposition to them, may also frequently lead to the formation of gall-stones.'[2] Lastly, the intimate relation between urinary calculi, biliary calculi, and gout was in accordance with the great clinical experience of the late Professor Trousseau,[3] and of late years has been strongly insisted on by his countryman, Dr. Sénac of Vichy.[4] In connection with these remarks it may be worth adding that, in a

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[1] Klinik der Leberkrank., Sydenham Society's translation, vol. ii. p. 511.

[2] 3rd edit. p. 369.

[3] Clinical Lectures, Sydenham Society's edit. vol. iv. p. 231.

[4] Coliques Hépatiques, Paris, 1870, p. 84.

usually after food, is loaded with albumen, whereas at others it contains none; and this state of matters may last for many months, and then completely disappear.[1] Now it is not contended that in all these cases the liver is the organ primarily at fault, but certainly in some there is good reason for believing it to be so, the albuminuria being unattended by any other symptom of renal disease, varying greatly in quantity and sometimes absent, and the urine being of normal quantity, of high specific gravity, and habitually loaded with lithates, lithic acid, oxalates, and pigments,[2] and there being very often cutaneous eruptions, dyspepsia, and other evidence of hepatic derangement. I have met with several instances of this sort where the patient was subject to severe attacks of what at first seemed to be hepatic colic, but where there was no jaundice and the paroxysm was followed by a temporary yet extraordinary increase of lithates and albumen in the urine. Lastly, so often have I observed albuminuria associated with hepatic disorder, which has disappeared completely and permanently when this has been set to rights, that I have little doubt that we have in the liver a cause of albuminuria to which attention has not hitherto been sufficiently directed.[3] The pathology of the albuminuria in these cases may be similar to that of certain cases of diabetes already referred to, the liver having too much work to do, and permitting some albumen to pass through in a form which cannot be assimilated; or possibly there may be some defect in the destructive functions of the liver, in consequence of which the albuminous matter, instead of being converted into urea, does not even reach the stage of lithic acid. It is possible that in many of the cases now referred to the albuminuria may indicate an early stage, not yet described, of the contracted or gouty kidney, yet it is certain that the symptom may persist or recur during many years without any probably *renal degeneration is a consequence of the long-continued elimination of products of faulty digestion through the kidneys.* I have seen this sequence of events so frequently, that I have no doubt as to their causative relationship. Dyspeptic symptoms, such as I have described, and consequent renal degeneration, are in some cases excited or greatly aggravated by habitual excess of alcohol.'[1] Numerous cases which have come under my own observation, and which I have carefully watched, have satisfied me as to the strict accuracy of Dr. Johnson's description; but the dyspepsia which so commonly precedes the first symptoms of contracted kidney is that which I have already described as accompanying persistent lithæmia, and as consisting in derangement, not so much of the gastro-intestinal digestion, as of the disintegrative processes which go on in the liver.

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[1] See some interesting cases of this sort recorded by the late Dr. J. Warburton Begbie, Edin. Med. Journ. April 1874. In a letter to Dr. Begbie respecting one of these cases, Dr. George Johnson observes: 'I have often met with cases in which the urine has been albuminous only after food and exercise.' In these cases both Dr. Begbie and Dr. Johnson coincided in thinking that the albuminuria was allied to the albuminuria of indigestion, and did not indicate structural disease of the kidneys.

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[3] Dr. W. Whitla has recently called attention to the influence of the liver in determining both anæmia and albuminuria. Dublin Journ. of Med. Science, February 1876.

Chronic degeneration of the kidneys with albuminuria is sometimes also a sequel of diabetes, the kidneys becoming diseased from the constant irritation of the saccharine urine. This is another way in which functional derangement of the liver may lead to structural disease of the kidneys.

There are also reasons for believing that albuminuria may be induced by hepatic derangement independently of structural disease of the kidneys. It is now generally acknowledged that albuminuria, even when copious, and in the absence of any acute febrile disorder, does not necessarily indicate renal disease. Very often in these cases the albuminuria is intermittent or remittent, and the albumen has peculiar chemical characters, the previous addition, for example, of a few drops of mineral acid preventing to an unusual extent the subsequent coagulability by heat.[2] Errors in diet are one of the most common causes. In some persons, peculiarly constituted, temporary albuminuria is a constant result of certain articles of food, such as uncooked eggs. In several instances I have known the urine passed at night to contain albumen, often associated with lithates and a high specific gravity, whereas the morning urine was clear, of low specific gravity, and contained no albumen. Again, in certain cases of exophthalmic goitre the urine at some hours of the day,

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[1] British Medical Journal, 1873, vol. i. pp. 161, 191.

[2] See Dr. Basham's work on Dropsy, 1863, also his work on Renal Diseases, 1870, p. 216; Dr. Leared, Med. Times and Gaz., Oct. 26, 1867; and P. Lorain, De l'Albuminurie, Paris, 1860, p. 57.

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other symptom of renal disease, and with but little impairment of the general health.[1]

6. *Structural Diseases of the Liver.*—It is highly probable that derangement of the disintegrative processes going on in the liver may lead to structural changes in the liver as well as in the kidneys. In the first place, fatty degeneration of the liver is well known to be a common lesion in persons who are large feeders or drink much alcohol and lead indolent lives, and in whose urine there are often copious deposits of lithates.[2] Under these circumstances, the change in the liver is sometimes associated with a similar change in the structure of the heart or kidneys, or with general corpulence. Again, in a large proportion of the cases of catarrhal jaundice, occurring towards middle or in advanced life, the patients have either previously suffered from actual gout, or they have been the subjects of lithæmia, with some of the symptoms of dyspepsia already described as accompanying it. Thirdly, although cirrhosis is most commonly the result of the direct irritation of the liver by alcohol, there are certain cases in which it cannot be traced to such a source, but in which its symptoms have been preceded for years by the lithic acid dyscrasia and dyspepsia. Cases of this sort have come under my own notice, and have been described by Baglivi, Stoll, Scudamore, and Trousseau as a 'chronic gouty hepatitis.'[3] Lastly, if we carefully investigate the antecedent medical history of patients suffering from primary cancer of the liver, we often find that for many years they have been 'bilious,' or they have been liable to dyspeptic symptoms attended by copious deposits of lithic acid or lithates in the urine, either habitually or from the slightest error in diet. That protracted derangement of cell-function should ultimately lead to abnormal cell-formation, in persons predisposed to cancer, does not appear to be an unwarrantable assumption.

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[1] Dr. Basham has communicated to me two such cases which have been under his observation, one for sixteen, and the other for ten years. In the latter the patient follows the active duties of the medical profession, and since he came under Dr. Basham's notice has married and become the father of four remarkably healthy children.

[2] Fatty liver is also very common in a very different condition of the system, viz. in wasting diseases, such as phthisis and chronic dysentery. It is supposed that the blood in these cases becomes charged with the oily matter which is rapidly absorbed from the patient's tissues, and that a portion of this becomes arrested in the liver.

[3] C. Scudamore, Nature and Cure of Gout, 4th edit. 1823, p. 43; Trousseau, op. cit. vol. iv. p. 381.

7. *Degeneration of Tissue throughout the Body.*—With the approach of old age there is a tendency of the tissues throughout the body to undergo degeneration and decay, fatty, and sometimes calcareous matter, being substituted for the normal structures. More than twenty years ago it was pointed out by Mr. Barlow,[1] that the 'climacteric disease' described by a former President of this College, Sir Henry Halford, and the 'Decline of the Vital Powers in Old Age' described by Dr. Marshall Hall, were in great measure due to these degenerations, and the fact is now generally acknowledged. In no organs are these degenerations more marked, or the cause of greater danger to life, than in the heart and in the coats of the arteries, the degenerations of which lie at the foundation of apoplexy, paralysis, aortic incompetence, and other maladies of advanced life. The explanation of these degenerations of tissue is to be found in derangements of the nutritive and disintegrative processes in which the liver plays so important a part; and in many instances, I do not say in all, it is the liver which appears to be primarily at fault. The nutrition of the tissues becomes impaired, partly perhaps in consequence of the supply of faulty nutritive material, but mainly from the functional activity of the liver becoming weakened with advancing age, as the result of which the blood and juices of the body are impregnated with a quantity of disintegrated albuminous matter, not sufficiently oxydised to be eliminated by the kidneys. But what in many persons is merely a form of senile decay may, under certain conditions, occur at a comparatively early period of life. Many observations have satisfied me that persons who habitually consume a large quantity of rich and stimulating food or of alcoholic drinks, who take little exercise, and whose urine is constantly loaded with an excess of lithic acid and lithates, are particularly prone to fatty degeneration. Andral and Lobstein long ago connected atheroma of the vessels with 'a particular taint of the fluids closely resembling gout,'[2] and it has been a common observation by physicians practising at spas resorted to by gouty patients, and borne out by my own experience, that atheroma of the arteries at an unusually early period of life, and diseases of the aortic valves which are not congenital and

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[1] General Observations on Fatty Degeneration, Medical Times and Gazette, May 15, 1852.

[2] C. E. Hasse, Diseases of the Organs of Circulation and Respiration, Sydenham Society's Translation, 1846, p. 82.

are independent of injury or rheumatism, are met with far oftener in persons who are the subjects of the lithic acid dyscrasia, or who have had gout, than in those who have no such tendencies.

8. *Local Inflammations.*—Lithæmia predisposes to local inflammations. Persons in good health, not apparently so, but in the strictest sense, are little liable to local inflammations. For example, when a given number of persons are exposed in common to an exciting cause of pneumonia, comparatively few are attacked with that disease; and, when the previous history of those attacked is investigated, it is usually found that, prior to the acute attack, they have been in an abnormal state of health. This was a point much insisted on by our late colleague Dr. Todd. In one of his remarkable clinical lectures, he writes as follows:—'Simple pneumonia is very rare in another sense also, that is, in its freedom from complication with, or dependence on, some peculiarity of constitution. To make this clearer to you, let me take an illustration. If two men, A and B, both in good health, be exposed to some noxious influence—cold, for example—at the same time, and for the same period, A will get a severe attack of pneumonia and B will not. Now, at first sight, one can scarcely conceive why the pneumonia should attack the one and not the other, for they were both apparently equally well at the time of exposure to cold; but, if we carefully examine into the previous history of these individuals, we shall find that A is of a gouty or strumous constitution, or has some peculiarity of diathesis which B does not possess, and it is by reason of this that A is seized with pneumonia when subjected to the noxious influence which produces no such injurious effect upon B.'[1] One of the conditions predisposing to local inflammations is gout, but the dyscrasia which I have designated lithæmia, and of which gout itself is only one of the local manifestations, acts in the same way. Having paid considerable attention to the matter, I am satisfied that persons with the lithic acid dyscrasia are much more prone than others to ordinary febrile colds, as well as to more severe local inflammations. They may appear robust and healthy up to the sudden occurrence of the inflammatory attack, but they have not really been in a normal state of health. I have also had occasion to observe that in certain persons who habitually pass an excess of lithates in the urine, the lithates cease to be

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[1] Clinical Lectures on Acute Diseases, 1860, p. 367.

certain symptoms, indicating derangement of the different physiological systems, but not constituting distinct diseases, which appear also to result from functional disorder of the liver.

IV. *Derangements of the Organs of Digestion.*

1. *The Tongue.*—It is well to remember that there may be considerable functional derangement of the liver and yet the tongue may be perfectly clean and normal, or at most only slightly coated in the morning; but in many cases, and particularly if the derangement be of old standing, the tongue presents the appearances commonly described as characteristic of 'atonic dyspepsia.' It is large, pale, and flabby, and indented by the teeth at the edge of the anterior third, while its surface is white, and the papillæ often elongated, so as to produce a pilous appearance. If the liver be somewhat congested, with these appearances we may often observe the fungiform papillæ on the tip and edges larger and redder than natural. In other cases, and especially when there is at the same time more or less gastric catarrh, the whole surface of the tongue is uniformly covered with a thick fur, sometimes whitish, but occasionally of a yellowish or brownish tint. According to Sir James Paget, psoriasis of the tongue, difficult to distinguish from syphilitic psoriasis, occasionally results from gout.[1]

2. *The Appetite* may be excellent, although there is great functional derangement of the liver with lithæmia, so that the patient is often tempted to eat what he knows from experience to disagree with him. But when the flow of bile into the bowel is deficient, the appetite is often bad, and there may be a loathing of fat and of greasy articles of diet. In cases which are not uncommon, where there is much functional derangement of the liver in conjunction with hepatic congestion and chronic gastric catarrh, there may be a loathing of all food excepting alcoholic stimulants, which increase the existing mischief, but which, as I have already stated, may lead to the accumulation of large quantities of fat.

3. *A Bitter Taste* is not unfrequently complained of by persons who are the subjects of jaundice; but the symptom is not due to the presence in the blood of bile-pigment, which is tasteless, but may be owing to its containing taurocholic acid,

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[1] Brit. Med. Journ. 1875, i. 737.

which is intensely bitter, or some abnormal product of disintegrated albumen. This may explain why many patients suffering from functional derangement of the liver, but who have not a trace of jaundice, often complain of a bitter, or sometimes a 'coppery,' taste in the mouth, especially in the morning.

4. *Dyspepsia.*—Flatulence is a common symptom of functional derangement of the liver. It is one of the most frequent results of a deficient flow of bile into the bowel, and when the bile-duct is completely obstructed it is rarely absent. It is likewise a common symptom in lithæmia, where there is often also a deficiency of bile in the bowel, and in all cases where the circulation through the liver is torpid. In all these cases, in consequence of a deficiency in the bowels of bile, which, as we have seen, is endowed with antiseptic properties, the intestinal contents undergo fermentation and gas is generated, which accumulates in the bowels, the distension being usually greatest from one to three hours after a meal. Acidity is another common symptom in lithæmic patients. Many articles of diet habitually disagree with such patients or render them 'bilious.' They awake next morning with a dry or clammy tongue, a bitter taste, frontal headache or vertigo, cramps, or pains in the knuckles.

5. *Constipation or Diarrhœa.*—In a large number of cases of functional derangement of the liver with lithæmia the bowels are more or less constipated; there is probably a deficiency in the quantity of bile which passes down the bowel, and therefore a want of the normal stimulus to peristaltic action. The motions are either unusually pale, or, from long detention in the bowel and the action upon them of the intestinal juices, they become black and lumpy. The latter condition is often associated with much depression of spirits, and hence the 'melancholia' of early writers on medicine.

But functional derangements of the liver may be attended by the opposite condition of diarrhœa, or constipation and diarrhœa may alternate. It is generally assumed that this diarrhœa is due to an increased secretion and discharge of bile; and, in fact, 'an excessive secretion of bile,' showing itself in 'copious, fluid, alvine evacuations, highly coloured with bile, often preceded by griping, by nausea, and sometimes by vomiting,' is one of the three functional derangements of the liver described by Dr. Copland[1] and other practical writers. Dr. Copland admitted that 'excessive biliary secretion is more frequently inferred from circumstances than proved by unequivocal evidence;' and, for my own part, without denying the possibility of the biliary secretion being sometimes excessive in quantity and unusually irritating, so as to excite diarrhœa, my experience has induced me to adopt a different explanation for the majority, at all events, of the cases of so-called 'excessive secretion of bile.' In most of these cases there is evidence of more or less congestion of the liver; the circulation through the liver is impeded, and there is a general stagnation of blood in the coats of the stomach and bowels. This mechanical stagnation is very likely to become converted into an active congestion or a catarrhal inflammation under the stimulus of irritating ingesta, so that even a small quantity of such a stimulus as alcohol may excite diarrhœa and vomiting. In many of these cases of 'bilious diarrhœa' the stools contain much mucus as well as bile. From what has been stated in my first lecture,[2] it is obvious that the large quantity of bile discharged from the bowel in these cases is no certain sign of an increased secretion by the liver, but may be due to a diminished absorption, consequent on irritation of the mucous surface.

6. *Vitiated Stools.*—A 'vitiated biliary secretion' is one of the three functional derangements of the liver described by systematic writers. Although there can be no doubt that the appearances of the bile in the gall-bladder after death are subject to great variations, too much importance has perhaps been attached to the characters of the stools during life as an index of the state of the liver. It must be remembered that variations in the characters of the stools may be the result of functional derangements or structural disease of the long tract of bowel between the entrance of the bile-duct and the anus, and may likewise depend on the rapidity or slowness with which this tract is traversed by the fæces. Bearing in mind these sources of fallacy, it may be said that when little bile is poured into the bowel, the stools are pale and unusually offensive, unless they be long delayed in the bowel, in which case they may be dark and lumpy; but when there is an excessive secretion or diminished absorption of bile, the motions are relaxed and liquid, and contain a much larger quantity of bile than in the normal state.

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[1] Medical Dictionary, ii. 725.

[2] Lect. XIV.

7. *Intestinal Hæmorrhage.*—Copious hæmorrhage from the bowels is well known to be an occasional result of cirrhosis and of other structural diseases of the liver which obstruct the portal circulation. I have frequently met with it, however, where there has been obvious derangement of the liver, but no reason to suspect structural disease. The patients have, for the most part, been beyond middle age; and I have known the attacks occur repeatedly in the same person at intervals of many years. The attack is usually preceded by a feeling of oppression and heaviness, or by creeping sensations and more rarely severe neuralgic pains suggesting gall-stones about the liver, by pain in the right shoulder, loss of appetite, nausea, and furred tongue; and the attack is often followed by a subsidence or cessation of these symptoms. Great relief is usually afforded by calomel or blue pill, with saline aperients. In the intervals of the attacks the patients may enjoy good health, except that they have to be careful as to diet. Not unfrequently they present the symptoms of the lithic acid dyscrasia, or they are subject to attacks of gout.

8. *Hæmorrhoids.*—In a large proportion of persons who suffer from hæmorrhoids the primary cause is in the liver. Hæmorrhoids are a common result of structural changes in the liver, such as cirrhosis; but they are also a frequent attendant of functional derangements, and especially of the loaded state of the liver so common in lithæmia.

9. *Hepatic Pain.*—With the exception just referred to, severe pain in the liver is not a common symptom of purely functional derangement of the organ. It may even be absent when there is advanced structural disease, unless there be inflammation of the peritoneal investment, or pressure upon a nerve by some morbid growth. But in cases of protracted lithæmia, a sensation of weight, fulness, tightness, or burning in the hepatic region is not uncommon; and when the bowels are neglected, or the patient continues to eat rich food and drink alcoholic stimulants, the liver is apt to become enlarged and congested and then it may be the seat of actual pain, which is usually increased after meals, or by lying on the left side.

10. *Jaundice.*—In considering whether jaundice may result from functional derangement of the liver, it will be necessary to enter somewhat in detail into its pathology. All cases of jaundice may be referred to one of two classes, viz.:—

(1) Cases in which there is a mechanical impediment to the

flow of bile into the duodenum, and where the bile is in consequence retained in the biliary passages, and thence absorbed into the blood.

(2) Cases in which there is no impediment to the escape from the liver into the bowel.

The several causes of jaundice belonging to each of these classes are given in the annexed Table. (See Lecture X., pp. 324, 334.)

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V. *Derangements of the Nervous System.*

1. *Aching Pains in the Limbs and Lassitude*, coming on about an hour after a full meal and sometimes associated with an irresistible tendency to drowsiness, are a very common symptom resulting from hepatic derangement with lithæmia. They are often attended by flatulence and other indications of atonic dyspepsia. In organic diseases of the liver, complaint is often made of a dull, heavy, or aching pain about the right, and more rarely about the left, shoulder-blade, which is accounted for by the connection existing between the branches of the subclavius nerve and the phrenic. Patients with lithæmia often complain of a similar pain.

2. *Burning or Scalding Patches* in the palms or soles, or in other parts of the body, are a common cause of complaint by gouty patients, as well as by those who are the subjects of lithæmia independent of gout. Sometimes the skin over the patch is slightly flushed; more commonly nothing abnormal is to be seen. These abnormal sensations may be persistent; but far oftener they are transient and frequently recur.

3. *Neuralgia.*—It is well known that gouty persons are very subject to sciatica, brachial neuralgia, and neuralgia in other parts. Indeed, as Sir James Paget has observed, a variously shifting neuralgia in a person of middle or advanced age, ought always to excite a suspicion of gout. The neuralgia in these cases is rapidly induced by errors in diet and is very common in the parts of the body which are the favourite seats of gout, such as the heel, the ear, the tongue, the palate, the fingers, or the breast.

In rare cases a neuralgic pain seems to occur in the liver itself. Many, probably most, of the reported cases of 'neuralgia hepatis' have probably been examples of biliary colic, where the stone has never advanced beyond the neck of the gall-bladder or the cystic duct, so as to cause jaundice. My experience would certainly lead me to doubt the purely neuralgic character of any such attack in which the pain is followed by jaundice, as has happened in some of the recorded cases. Other instances of supposed hepatic neuralgia have probably been examples of nephritic colic from renal calculi, where the pain, as I have known happen, radiates horizontally forwards, instead of taking the usual direction downwards towards the pubes. But, making allowance for such mistakes in diagnosis, a certain number of cases remain, which appear to be examples of true neuralgia of the liver. Cases of this sort have been described by Trousseau,[1] Anstie,[2] and other authors, although they have not come under my own notice (p. 485). The patients in these cases have been liable to sudden attacks, often periodic, of severe pain in the right hypochondrium, and radiating thence to the right shoulder, with tenderness over one or more of the dorsal spinous processes. They have been for the most part of nervous temperament, and subject to neuralgic pains in other parts of the body; and in most instances the attacks have been attended with great depression of spirits. The cause of the attacks is to be sought for in a general neuralgic tendency rather than in any disorder of the liver. Trousseau, however, has pointed out that hepatic colic from gall-stones may excite a true neuralgia. After showing how the peripheral irritation of a false tooth may excite facial neuralgia, he observes: 'The same thing obtains in hepatic colic. Fearful pain sets up suddenly in the pit of the stomach and in the region of the gall-bladder and of the ductus communis choledochus. So far, there is merely local pain, without neuralgia, and there is no tenderness on pressure of the dorsal spinous processes; but after two or three days spent in acute pain, a sharp pain is frequently complained of in the seventh, eighth and ninth intercostal spaces, in the shoulder, in the neck, and in the arm on the same side; from that time, neuralgia exists, and the vertebræ become very tender on pressure.'[3]

4. *Severe Cramps* in the legs, abdomen, and in different parts of the body are common in persons who are the subjects of lithæmia. They often come on during the night, and they are most common in cold and damp weather. The late Sir

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[1] Clinical Lectures, Sydenham Society's Translation, iv. 236.

[2] Anstie on Neuralgia, 1871, p. 61.

[3] Op. cit. Sydenham Society's edit. i. 482.

Charles Scudamore remarked that in some gouty persons they produced such intense suffering, as to form the leading feature of the disease.[1] Sometimes they precede a paroxysm of articular gout. Two remarkable instances of this result of lithæmia have been recorded by Dr. Bence Jones. The first was that of a gentleman aged 40, who for years had been liable to constant deposits of lithic acid and lithates in the urine. He then became the subject of attacks of violent pain in the stomach, coming on from one to five hours after a late dinner. The pain was intermittently spasmodic; the most intense pain was reached in half a minute; it then relaxed, and returned as badly as before in two minutes. His suffering lasted about an hour, when the pain gradually abated, leaving a tenderness on pressure and an irritability after food for two or three days. After the attack, the urine always deposited lithic acid crystals. These attacks had lasted for several months, but, under a careful diet and the use of alkalies, they entirely ceased. The second patient, who also was the subject of lithæmia, was seized with violent cramps in the rectum, coming on six or eight hours after food, and lasting from half an hour to an hour. The attacks entirely ceased under the same treatment as in the first case.[2]

5. *Headache* is a not unfrequent result of hepatic derangement. Most commonly it takes the form of a dull heavy pain in the forehead, more rarely in the occiput, complained of as soon as the patient awakes in the morning, and either speedily ceasing or lasting the greater part of the day or for several days. Such headaches are common in the subjects of lithæmia after any indiscretion in diet, or when the bowels are constipated. Their immediate cause is probably the presence in the blood of some abnormal product of albumen-metamorphosis; the derangement of the liver is usually indicated by pain and fulness in the right hypochondrium, flatulence, and high-coloured urine loaded with lithates; and relief is usually afforded by mercurial and saline purgatives and alkaline diuretics.

From these headaches it is necessary to distinguish *Megrim*, which unfortunately is the form of headache to which the terms 'bilious' or 'sick' are still popularly applied. This is a neuralgia, which probably in many cases is in no way connected with hepatic derangement, the bilious symptoms being the result, and not the cause of the attack, and the presence of bile in the vomited matter being, as in sea-sickness, simply due to the urgency of the vomiting. Although this view was ennunciated two centuries ago by Sydenham, and since his time has been clearly set forth in many medical writings, of which I would mention, in particular, the Gulstonian Lectures of the late Dr. Symonds, delivered in this College in 1858, and the excellent work of our associate Dr. Edward Liveing,[1] recently published, it is still the fashion to attribute these attacks to 'biliousness,' or to 'an excess of bile in the system.'

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[1] Nature and Cure of Gout, 4th edit. 1823, p. 532.

[2] Lectures on Pathology and Therapeutics, 1867, p. 85.

But, while fully admitting that megrim is in no way connected with retained bile, I agree with those authors who believe that certain cases of megrim are toxic in their origin, being symptomatic of gout and of some other disorders. The late Sir Henry Holland, in his 'Medical Notes and Reflections,' described hereditary periodic headaches associated with gout, and he added: 'In conformity with this view, there is reason to believe that the kidneys are the excretory organs most concerned in giving relief in these cases, and principally by an increased separation of lithic acid and its compounds.'[2] Megrim, as Dr. Liveing states, is sometimes the expression of what is called a latent gouty diathesis, or, in other words, of lithæmia. A father may have suffered from gout, and his son may become the victim of megrim. In some patients megrim terminates when they are attacked with gout. 'So evidently,' observes Trousseau, 'is it (megrim) a manifestation of the gouty diathesis, that articular gout and megrim are observed in the same person, the one subsiding on the appearance of the other; and that it is often also the only expression of the hereditary tendency in subjects who are the children of decidedly gouty parents.'[3] In connection with these observations, I would call attention to certain cases which have come under my notice of severe neuralgic headache occurring in connection with contracted granular kidneys, and being sometimes the first symptom for which the patient has sought medical advice. The headache in these cases also was evidently toxic, and in one instance fatal coma followed the subcutaneous injection of a quarter of a grain of morphia. The headache has been so severe that more than once I have known the case diagnosed as one of cerebral tumour. I have met with these cases so frequently, that I believe it to be a good rule to investigate the condition of the kidneys in all cases of neuralgic headache occurring for the first time in persons of middle or advanced age, before having recourse to treatment.

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[1] On Megrim and some Allied Disorders, London, 1873.

[2] Medical Notes and Reflections, 1839, p. 288.

[3] Op. cit. Sydenham Society's edit. iv. 378.

In this way, then, megrim may sometimes be traced to hepatic derangement, this derangement consisting, not in the retention of bile, but in that condition of liver which we have found to produce lithæmia and often to lead to gout; and, in accordance with this view, I have often noticed that megrim has been produced by particular articles of diet and relieved by mercury, podophyllin, and other remedies which unload the liver.

6. *Vertigo, Temporary Dimness of Sight, Double Vision, &c.*—Sudden attacks of giddiness are in many instances similar in their pathology to megrim, and giddiness in certain patients replaces the neuralgia. But giddiness, often associated with specks or waviness before the eyes or flashes of light, or double vision, according to my experience, is in a much larger proportion of cases connected with hepatic derangement, lithæmia, and gout, and follows the use of certain articles of diet, such as tea, champagne, citron, etc. Many years ago, Boerhaave's commentator related the case of a man, who during two years was always seized with vertiginous symptoms when he attempted to stand up. In vain had the ablest practitioners endeavoured to cure him. Quite suddenly he had an attack of gout, of which disease up to that date he had had no indication; and from that moment he found himself free from the vertigo to which he had formerly been liable.[1] A medical friend of my own who has long suffered from gout, as certainly as he drinks a cup of tea or a glass of champagne, is seized, often while walking in the street, with sudden giddiness; his head feels empty, and neighbouring objects seem to whirl round him; he does not lose consciousness, but he would fall did he not lay hold of the railing. After a few seconds or minutes the attack passes off, but in some patients it is more persistent. Another friend, who never has had gout, but whose urine is frequently loaded with lithates, was seized with dimness of sight and giddiness every night while writing. He took iron, quinine, and other tonics, but he got worse instead of better. He was advised to give up his profession for a time

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[1] Trousseau, op. cit. Sydenham Society's edit. iv. 379.

and try the effect of change of air; but, before taking so serious a step, he took a few doses of blue pill, and the symptoms at once and permanently disappeared. A third patient under my care, who for years had been subject to lithæmia, but never had gout, would be suddenly seized, while writing, with dimness of sight and specks floating before the eyes, or even with complete, but temporary, blindness of one eye. Here also iron and quinine disagreed, but the symptoms were removed by remedies directed against the liver. Many writers have referred attacks such as those which I have now described to derangements of the stomach. Trousseau, for example, who has described them under the designation of 'vertigo à stomacho læso,'[1] speaks of them as associated with epigastric pain increased by food, flatulence, acid eructations, and vomiting of glairy mucus; but he admits that the gastric derangement in which the vertigo is supposed to originate may not show itself, and this admission certainly accords with my experience. On the other hand, the circumstance of the frequent association of the vertigo with gout or lithæmia, and the fact that alkalies and aperients, which are the best remedies for these conditions, are also the remedies most likely to prevent a recurrence of the attacks of vertigo, make it probable that this has a toxic origin and that the liver is the organ mainly at fault.

7. *Convulsions.*—In January of the present year (1874) I saw a gentleman, about 58 years of age, suffering from cirrhosis of the liver. He had all his life been addicted to the pleasures of the table, and had suffered from hepatic derangements as long as he could remember. Six years ago, he became subject to severe spasmodic twitchings in his legs, followed on three occasions by several epileptiform seizures. Shortly after the last fit he had his first attack of gout, and since then he had suffered repeatedly from the gout, but there had been no return of the convulsions or of the muscular twitchings. There was no evidence of renal disease. Many similar cases are, I believe, on record. For example, Van Swieten mentions the case of a man who had violent abdominal pains accompanied by delirium and general trembling, and subsequently a severe attack of epilepsy. From that date he had repeated attacks of gout, but no return of the nervous symptoms.[2] Several similar cases are related by Garrod. One gentleman had been liable to frequent

[1] Op. cit. Sydenham Society's edit. iii. 537.

[2] Ib. iv. 379.

epileptic attacks from 20 to 52 years of age. He then had for the first time a severe attack of gout in one big toe; from 52 to 92, when he died, he had frequent recurrences of gout, but no epileptic attacks. In another case of 'gouty epilepsy' Dr. Garrod found lithic acid in the blood.[1]

8. *Mania*, like epilepsy, is an occasional result of lithæmia, ceasing suddenly and permanently on the advent of an attack of gout.

9. *Paralysis.*—I have repeatedly met with patients who have complained of numbness, tingling and pricking sensations—the feeling as if the part were asleep—or a feeling of coldness or creeping in the extremities on both sides, or only on one. These symptoms may last for months or years, and may be associated with headache, nausea, and depression of spirits. They often cause needless alarm by exciting the suspicion that paralysis is imminent; but, if associated, as they often are, with lithæmia, oxaluria, or other evidence of hepatic derangement, they may entirely and permanently disappear under the use of calomel, saline aperients, alkalies, and attention to diet.

10. *Noises in the Ears* are common symptoms in gout[2] and also in lithæmia independent of gout. One patient has the feeling of a strong wind blowing into the ear; another compares the noise to that of flowing water, or of singing or buzzing; while in another the sounds have a pulsating character, the sounds corresponding to those of the heart.

11. *Sleeplessness* may, of course, arise from many different causes, but one of its causes is that derangement of the liver which produces lithæmia. When this is the case, the patient is often heavy and drowsy after a full meal, and he may fall asleep at once on retiring to rest; but, after one, two, three, or four hours, he awakes, and then he either lies awake for hours, or he is constantly falling asleep, dreaming, or having the nightmare and awaking—four or five times or even oftener in the course of one hour—until the morning comes, when he drops into a quiet sleep of an hour or more, or he is obliged to get up tired and irritable. This sleeplessness, like the vertigo we have already considered, is often induced by particular articles of diet, or by some unwholesome combination of them. What will excite headache, giddiness, or disorders of the circula-

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[1] On Gout, 3rd edit. 1876, p. 460.

[2] Scudamore, op. cit. p. 376.

tion in some patients will in another cause sleeplessness. Sometimes, however, this symptom will occur when the patient is most careful as to diet. What is important also to note is, that in most of these cases there are no obvious symptoms of gastric dyspepsia; the appetite may be good—too good, in fact; the bowels may be regular; and there may be no pain, flatulence, or other discomfort after meals; but there will be found an unusual tendency to the deposit of lithates in the urine, and very often other phenomena of a so-called gouty diathesis. This form of sleeplessness was described a century ago by Cullen, the distinguished nosologist, in these words: 'Persons who labour under a weakness of the stomach, as I have done for a great number of years past, know that certain foods, without their being conscious of it, prevent sleeping. So I have been awakened a hundred times at two o'clock in the morning when I did not feel any particular impression; but I knew that I had been awaked by an irregular operation in that organ, and I have then recollected what I took at dinner, which was the cause of it. Dr. Haller is liable to the same complaint; and in his larger work especially, he gives the particulars of his own case.'[1] The affection has also been well described by Dr. Dyce Duckworth in some excellent observations on different forms of sleeplessness recently published.[2] It is, however, a form of sleeplessness not generally understood, and harm is often done to patients suffering from it by the administration of opiates and other soporifics, in ignorance of its real cause. Very often the symptom will be greatly relieved, if not entirely removed, by careful attention to diet, and particularly by moderation in, or abstinence from, wine; and, in some cases, a dose of carbonate of soda when the patient goes to bed, or when he first awakes, is of service. Some patients with this form of sleeplessness have told me that they never sleep so well as after a dose of calomel or blue pill.

12. *Depression of Spirits.*—The influence of the liver upon the animal spirits has been recognised by medical writers in all ages. To the belief in the existence of such an influence may be traced the origin of such terms as *Hypochondriasis* and *Melancholia*. Although it is not contended that the morbid states of mind, to which at the present day we apply these terms, have their origin in the liver, they are unquestionably

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[1] Institutions of Medicine, 1770.

[2] British Medical Journal, December 27, 1873.

in many instances, accompanied and aggravated by derangements of this organ; and it is equally true that, independently of either hypochondriasis or melancholia, persons with functional derangement, or structural disease, of the liver are subject to fits of great depression of spirits and often groundless fears of impending danger, which cease when the liver is restored to its normal state.

13. *Irritability of Temper* is another common symptom of functional derangement of the liver, and is sometimes the first indication of anything wrong. A man who has previously borne the crosses of life with equanimity and been amiable to those about him gradually becomes disconcerted by trifles; his mind broods upon them; and he makes all around him unhappy and himself the most miserable of all. His relatives perceiving no other sign of indisposition, and failing to recognise the true cause, too often put down the ebullitions of temper to something mentally or morally wrong, to moral depravity, or failure to make any mental effort; but remedial measures calculated to restore the liver to healthy action, if resorted to in time, will often remove the irritability, and either the patient's improvement under such treatment, or an attack of gout, reveals the cause of the patient's bad temper. In his 'Psychological Inquiries,' the late Sir Benjamin Brodie thus speaks of a patient with a superabundance of lithic acid in the blood: 'Uncomfortable thoughts are presented to his mind: he becomes fretful and peevish, a trouble to himself, and, if he be not trained to exercise a moral restraint over his thoughts and actions, a trouble to everyone about him. After a while, the poison, as it were, explodes: he has a severe attack of gout in his foot; he is placed on a more prudent diet; the system is relieved of the lithic acid by which it was poisoned. Then the gout subsides; happy and cheerful thoughts succeed those by which the patient was previously tormented, and these continue until he has had the opportunity of relapsing into his former habits, and thus earning a fresh attack of the disease.'[1]

14. *Cerebral Symptoms and the Typhoid State.*—It is well known that restlessness, delirium, stupor, coma, subsultus, tremors, convulsions, a dry, brown tongue, and other phenomena of the 'typhoid state,' are apt to supervene in certain cases of advanced disease of the liver, whether attended by jaundice or not. These symptoms have been usually attributed to a sup-

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[1] Second edit. 1855, p. 73.

pressed secretion of bile. But the assumption that the elements of the bile are preformed in the blood, and are merely separated from the blood by the liver, we have already found to be devoid of foundation; and we have also found that bile is far from being, as commonly supposed, a deadly poison, and that its presence in the blood, even to saturation, does not give rise to cerebral symptoms. The cerebral symptoms referred to are often most severe when the jaundice is slight, or when there is none; and they are readily accounted for by the knowledge of the disintegrating function which the liver is now known to perform. When this function of the liver is arrested or seriously impaired, urea is no longer eliminated in sufficient quantity by the kidneys, lithic acid and deleterious products of disintegrating albumen even less oxydised, such as leucin and tyrosin and perhaps others with which we are as yet imperfectly acquainted, accumulate in the blood and tissues; and the result is the development of symptoms of blood-poisoning similar to those which arise when the kidneys are unable to eliminate the products of albumen-degeneration owing to disease of their own structure, or to an excessive formation of urea and other products as happens in many febrile diseases. In acute atrophy, for example, the structure of the liver is destroyed and its functions arrested; leucin and tyrosin take the place of urea in the urine and are also found in large quantity in the liver, spleen, and kidneys; while cerebral symptoms and the typhoid state are prominent features of the disease.

LECTURE XVI.

THE CROONIAN LECTURES ON FUNCTIONAL DERANGEMENTS OF THE LIVER.

VI. DERANGEMENTS OF THE ORGANS OF CIRCULATION. 1. PALPITATIONS AND FLU TERINGS OF THE HEART; 2. EXAGGERATED PULSATION OF THE LARGE ARTERIES; IRREGULARITIES AND INTERMISSIONS OF THE PULSE; 4. FEEBLE CIRCULATION; ANÆMIA; 6. ANGINA PECTORIS; 7. VENOUS THROMBOSIS.—VII. DERANGEMENTS ORGANS OF RESPIRATION. 1. CHRONIC CATARRH OF FAUCES; 2. BRONCHITIS; SPASMODIC ASTHMA.—VIII. DERANGEMENTS OF THE GENITO-URINARY ORGANS. DEPOSITS OF LITHIC ACID AND LITHATES IN URINE; 2. RENAL CALCULI; 3. DISEAS OF KIDNEYS; 4. CYSTITIS; 5. URETHRITIS; 6. CHORDEE; 7. ORCHITIS.—IX. ABNORMA CONDITIONS OF THE SKIN. 1. ECZEMA, LEPRA, PSORIASIS, AND LICHEN; 2. URTICARIA 3. BOILS AND CARBUNCLES; 4. PIGMENT-SPOTS; 5. XANTHELASMA; 6. PRURITUS.— C. CAUSES OF FUNCTIONAL DERANGEMENTS OF THE LIVER.—I. SECONDARY. 1. STRUCTURA DISEASES OF THE LIVER; 2. DISORDERS OF STOMACH AND BOWELS; 3. DISEASES THE HEART AND LUNGS; 4. PYREXIA.—II. PRIMARY.—1. ERRORS IN DIET; 2. DEF CIENT SUPPLY OF OXYGEN; 3. HIGH TEMPERATURE; 4. NERVOUS INFLUENCES; CONSTITUTIONAL PECULIARITIES; 6. POISONS.—D. TREATMENT OF FUNCTIONAL D RANGEMENTS OF THE LIVER. 1. DIET; 2. FREE SUPPLY OF OXYGEN; 3. DILUENTS 4. BATHS; 5. APERIENTS—CHOLAGOGUES; 6. ALKALIES; 7. CHLORINE, IODINE, BROMIN AND THEIR SALTS; 8. MINERAL ACIDS; 9. TONICS; 10. OPIUM. CONCLUDING REMARKS

Mr. President, Fellows of the College, and Gentlemen, —In my last lecture I considered some of the more importan diseases and symptoms resulting from abnormal disintegratio of albuminous matter in the liver. I have still to refer to certain derangements of the organs of circulation and respiration, and to abnormal states of the skin traceable to the same cause. I shall then mention some of the chief causes of functional derangements of the liver, and conclude the lecture by a brief sketch of the principal rules for treatment of these derangements.

VI. *Derangements of the Organs of Circulation.*

1. *Palpitations and Flutterings of the Heart.*—Indigestion has long been regarded as one of the causes of palpitation independent of organic disease of the heart. Many patients with

this functional derangement of the heart describe their sensations as that of a transient fluttering rather than a continuous palpitation; and when this feeling of fluttering comes to be investigated, it usually turns out to be produced by a strong thump of the apex of the heart following one or more weaker beats or a decided stop. In some of these cases of palpitation and fluttering a prominent symptom of the indigestion is flatulence; and then the common explanation of the cardiac symptoms is, that they are due to the pressure upon the heart of the distended stomach and bowels; and this explanation receives support from the fact that, on the removal of the flatulence, the cardiac symptoms are often relieved or cease. But in other of these cases the flatulence may be entirely removed, while the cardiac symptoms remain; while in others there is not the slightest evidence of flatulence, and still the cardiac symptoms are removed by remedies, such as alkalies and aperients, calculated to improve the condition of the liver. It seems probable, therefore, that in some, if not in many, cases, when flatulence and palpitations coexist, they do not stand in the relation of cause and effect, but are both the result of a common cause. Palpitations and still more flutterings of the heart are particularly common in gouty people, whether they suffer from dyspeptic symptoms or not. Everyone present must have met with cases of the sort. Scudamore relates cases in which patients suffered from severe palpitations for six months without any relief from medicine; but, on the occurrence of a fit of gout, the palpitations suddenly and entirely ceased.[1] Dr. Garrod, in his work on Gout, remarks: 'One of the most common symptoms produced by a gouty state of the system is palpitation of the heart, often accompanied by irregularity of its rhythm, and occasionally with pulsation of some of the larger arteries. In the majority of these cases, the condition is secondary to dyspepsia, but at times it may be directly excited by the impure condition of the blood; and I have notes of some cases in which no organic mischief could be discovered in the heart, nor any sign of indigestion, and the symptoms ceased on the occurrence of gout in the joints.'[2] These cardiac symptoms are also very common in persons who are the subjects of lithæmia or oxaluria, but who never have gout. They are often the first symptoms to draw the patient's notice to the fact that his health is not what it ought to be; they cause great depression of spirits; and very often they are aggravated by injudicious treatment, and especially by the use of iron, which may seem to be indicated by the patient's anæmic aspect, but which is rarely tolerated until the liver has been brought into a healthy state by alkalies, aperients, and attention to diet. Although in the cases now referred to the cardiac symptoms result from pneumogastric irritation by a poison in the blood, the fact already referred to must not be lost sight of, that this same morbid state of blood may ultimately lead to degeneration of the muscular wall of the heart, or to disease of the aortic valves (see p. 576).

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[1] Op. cit. pp. 16, 98, 374.

[2] Nature and Treatment of Gout, 1859, p. 510.

2. *Exaggerated Pulsation of the Large Arteries.*—Dr. Matthew Baillie, in a communication made to this College on December 2, 1812, was the first to call attention to cases of increased pulsation of the aorta in the epigastric region, simulating aneurism, but in some instances lasting twenty-five years or longer, and the result merely of 'imperfect digestion with an irritable constitution.'[1] This exaggerated pulsation, not only of the aorta, but of other arteries, independent of either contracted kidney or aortic regurgitation, is now well known, and one cause of it appears to be a morbid state of blood resulting from derangement of the liver and often associated with gout. Scudamore relates cases of palpitations in the head occurring in persons afflicted with bilious derangement and gout, and likewise the case of a gentleman who had gout and bilious derangement, and who suffered alternately from palpitation of the heart and pulsation of the aorta in the epigastric region.[2] Garrod also speaks of an irritable state of the aorta and pulsation of the larger arteries as occasionally resulting from gout.[3] The undue pulsation in these cases is often subdued by treatment directed against the liver.

3. *Irregularities and Intermissions of the Pulse.*—An intermitting pulse, which may, or may not, be attended by the sensation of fluttering of the heart already referred to, results from a variety of causes, of which the principal are these:—

a. Valvular and other diseases of the heart. In organic diseases of the heart, however, irregularity of the rhythm is more common than decided intermission.

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[1] Medical Transactions, published by the College of Physicians, 1813, vol. iv. p. 274.

[2] Op. cit. p. 98.

[3] Op. cit. pp. 510, 511.

b. A weakened or unduly irritable state of the nervous system, such as that which often occurs in old age, or sometimes appears to be constitutional, or those which are induced by fevers, delirium tremens, hysteria, protracted want of sleep, anxiety, etc.

c. Morbid states of the blood associated with gout or lithæmia, or with other evidence of hepatic derangement. It has been the fashion to attribute the intermission in these last cases to pneumogastric irritation by gastric dyspepsia or flatulence ; but, just as we found in vertigo and palpitation, there is very often neither flatulence nor other evidence of gastric derangement; and my experience has led me to the conclusion that in most of these cases the pneumogastric irritation has a toxic origin, or is due to the presence in the blood of some morbid material resulting from derangement of the liver. What this material is we do not with certainty know. It is not uncommon for the pulse to become very slow, or even to be irregular or to intermit, in jaundice. These symptoms do not appear to be due to the presence of bile-pigment in the blood, for in many cases of jaundice they are absent; but the experiments made by Rohrig, Legg, and others have shown that the bile-acids paralyse the heart and retard its action, while bile-pigment has no such effect.[1] It is possible, then, that slowness and intermission of the pulse may be caused by the presence in the blood of unchanged bile-acids, even in cases where there is no jaundice; but probably another cause of the intermission is some product of albuminous disintegration, inasmuch as it is so commonly met with in connection with lithæmia or gout, and as it is often entirely removed by blue pill, saline aperients, alkalies, and attention to diet. A notable fact in these cases is that the tendency of the pulse to intermit is usually greatest when the patient is at rest, and is diminished or ceases on his taking exercise. As in the case of vertigo or sleeplessness, the intermission may be excited by particular articles of diet. It may last for many years, during which the patient may enjoy very fair health and be capable of considerable exertion. I lately saw a gentleman, aged eighty, who had had an intermitting pulse for upwards of fifty years. He had suffered from gout and dyspepsia, but in his eightieth year he could walk long distances and up moderate ascents without difficulty. It is also worth noting that intermission of the pulse may last for years, and then entirely disappear. Dr. C. Lasègue of Paris, who has published an interesting memoir[1] upon intermitting pulse, thinks that it is chiefly met with under two conditions, viz.: firstly, as an accompaniment of some chronic general morbid state, which is the prelude of some more acute mischief, on the development of which the intermissions may cease; and secondly, as the accompaniment of a general morbid state consequent on the first establishment of some local disease, the general cachexia and the intermitting pulse after a time disappearing, although the primary local disease remains. The following case, communicated to me by Mr. Paul Jackson, is a remarkable illustration of the complete disappearance of the intermission after a duration of several years, and also of its toxic origin.

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[1] See Lecture IX., p. 320.

About the year 1838, Mr. J. T., then forty-two years of age, of nervous temperament, a generous liver, and subject to hepatic derangement, began to suffer from intermission of the pulse and a fluttering sensation at the heart. He had no dyspnœa nor other symptom of cardiac disease, and there was no abnormal cardiac murmur. He saw a great many physicians, but got no relief; but, after upwards of three years, he had a severe attack of urticaria, whereupon the intermission and the fluttering entirely and for ever ceased. He lived for twenty years afterwards, and, with the exception of occasional attacks of gout and of sudden vertigo, he enjoyed good health. He died at last, however, suddenly, of rupture of the heart, at the age of sixty-five.

It may be well to add that, even when intermitting pulse coexists with valvular disease of the heart, it appears to be sometimes due to hepatic derangement rather than to the cardiac lesion. Take, for example, aortic incompetence. The rhythm of the pulse in this lesion is usually regular, but in rare instances it is irregular and intermitting. From the supervention of this symptom the patient's condition is often believed to have become more perilous, and yet there may be no aggravation of the other cardiac symptoms. The pulse may become regular, instead of more intermitting, after exercise, and the intermission may be entirely removed by the same remedies as are effectual when there is no cardiac disease. That the intermission of the pulse should be independent of the cardiac lesion is not surprising, when we remember that atheroma of the arteries, which is the main cause of aortic incompetence occurring in middle or advanced life,[1] and intermitting pulse may both result from the lithic acid dyscrasia. The following case is an illustration of what I have just stated.

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[1] Des Intermittences Cardiaques, par le Dr. C. Lasègue, Arch. Gén. de Méd. December 1872.

A discharged soldier, aged 56, came under my care in July 1873 for what appeared to be muscular or neuralgic pains. He was found to have aortic incompetence, but he had never had any symptom of cardiac disease—pain, palpitation, or dyspnœa—and he had been employed as a porter in a public institution, one of his duties being to carry heavy coal-scuttles up long stairs, from which he had apparently suffered no inconvenience. His pulse was quite regular. He was treated with quinine, but he left the hospital after some weeks not much relieved, and returned to his work. In December he again came to me, complaining of pain in his right shoulder and constipation; his pulse was now very intermitting, and he had also a frequent feeling of fluttering about his heart. He had no other cardiac symptom, and on walking his pulse became regular. He was now treated with blue pill, colchicum, aperients, alkalies, and iodide of potassium, and in a short time the pain in the shoulder, the fluttering, and the intermission disappeared.

4. *Feeble Circulation.*—In cases of protracted hepatic derangement, symptoms of feeble circulation, which may be independent of palpitations or irregularities of the pulse, are not uncommon. The patient complains of languor, debility, and coldness of the extremities. The heart is found to beat feebly, but to be free from organic disease; there is evidence of hepatic derangement, and the urine often deposits lithates. Iron, quinine, and alcoholic stimulants, which are frequently prescribed for this condition, may render the patient worse instead of better, and the surest way to increase the strength of the heart is to avoid alcohol and relieve the liver.

5. *Anæmia* (see page 579).

6. *Angina Pectoris.*—The neuralgic affection known as angina pectoris probably arises in many different ways. One cause appears to be the lithic acid dyscrasia. It is now many years since an English physician, Dr. William Butter, described certain cases of this affection as diaphragmatic gout. The patients had been careless as to diet, and 'particularly fond of

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[1] I do not remember to have met with intermitting pulse in cases of aortic incompetence of rheumatic origin.

the stronger malt liquors'; the urine deposited 'a copious gross sediment'; and the attack might terminate in a fit of the gout.[1] Many writers have since then described a 'gouty cardialgia'; and Trousseau has pointed out that certain cases of angina pectoris are independent of any disease of the heart or great vessels, and are merely a 'manifestation of the gouty diathesis.'[2] Not long since, I saw a gentleman, aged 65, who complained of awaking in the night three or four times a week with violent pain in the cardiac region, extending up to the left shoulder and down the left arm. I could discover no sign of disease in his heart. He stated that, six years before, he had suffered for months from similar attacks, but had recovered under medical treatment. He had never had gout, but he was very careless as to his diet, and his brother I knew to be a martyr to gout. Under the use of alkalies and blue pill the angina again disappeared.

7. *Venous Thrombosis.*—There are good reasons for believing that the morbid condition of blood resulting from functional derangement of the liver not unfrequently leads to the production of venous thrombosis. Cases of this sort have been described as 'gouty phlebitis' by Sir James Paget, who observes:—

> 'The use of this name is, I believe, justified by the number of cases in which phlebitis is associated with ordinary gouty inflammation in the foot or joints, and occurs, with little or no evident provocation, in persons of marked gouty constitution, or with gouty inheritance. In such cases the phlebitis may have no intrinsic characters by which to distinguish it; yet, not rarely, it has peculiar marks, especially in its symmetry, apparent metastases, and frequent recurrences. Gouty phlebitis is far more frequent in the lower limbs than in any other part; but it is not limited to the limb that is, or has been, the seat of ordinary gout. It affects the superficial rather than the deep veins, and oftener occurs in patches, affecting (for example) on one day a short piece of a saphenous vein, and on the next day another separate piece of the same, or a corresponding piece of the opposite vein, or of a femoral vein. It shows herein an evident disposition towards being metastatic and symmetrical; characters which, I may remark, by the way, are strongly in favour of the belief that the essential and primary disease is not a coagulation of blood, but an inflammation of portions of the venous walls. The inflamed portions of vein usually feel hard, or very firm; they are painful, aching, and very tender to the touch; such pain, indeed, often precedes the clearer signs of the phlebitis, and

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[1] Treatise on Angina Pectoris, 2nd edit. London, 1806.

[2] Op. cit. vol. iv. p. 379.

not rarely begins suddenly. The integuments and the affected veins (where they are superficial) are slightly thickened and often marked with a dusky reddish flush. When superficial veins alone are affected there may be little œdema; but when venous trunks, as the femoral, the whole limb assumes the characteristics of complete venous obstruction. It becomes big, clumsy, featureless, heavy, and stiff; its skin is cool and may be pale, but more often it has a partial slightly livid tint, with mottling from small cutaneous veins visibly distended. The limb thus enlarged feels œdematous all through, but firm and tight-skinned, not yielding easily to pressure, and not pitting very deeply. By this state alone the disease must sometimes be recognised, for it may be very marked when only a small portion of vein is affected, and that (as the lower part of the popliteal) so deeply seated as to be scarcely felt. The constitutional disturbance associated with this condition is that of slight feverishness, or of an ordinary gouty attack, more or less acute in different cases. The effects of the disease I have never had an opportunity of examining by dissection, for in the only fatal case that I have seen no autopsy was allowed. So far as one may judge of them by after-events during life, the veins which may have been obstructed become in some cases pervious again; for in some cases the clearing up of the œdema and the restoration of the healthy condition of the limb are complete, yet the veins remain apparently very susceptible; they ache exceedingly during fatigue or trivial illness, or in changing weather; and I have known phlebitis excited by trivial causes in the same veins three or four times. In other instances, however (but I think they are rarer than in other forms of phlebitis), the obstruction of the veins appears complete and permanent; and then, if they be trunk-veins, the limb remains permanently enlarged, cumbrous, and heavy. Its superficial veins may after some time become varicose, and others may enlarge for collateral blood-streams; and I believe that an increased growth may take place in some of the tissues, especially the muscles of the limb.'[1]

As in other forms of thrombosis, so here, the clot may become broken up and its fragments be dispersed, and in this way syncope, or even sudden death, may result from embolism of the pulmonary artery. Like gout, this form of thrombosis is often hereditary; but it is well to remember that it may be induced by functional derangement of the liver in persons who neither inherit gout, nor have at any time had traces of it themselves.

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[1] On Gouty and some other forms of Phlebitis, St. Bartholomew's Hosp. Rep. 1866, vol. ii. p. 83.

6. *Chordee.*—Persistent, and sometimes painful, erections of the penis during sleep occasionally result from lithæmia, especially in elderly persons. Even in persons of middle age I have known them to be a cause of constant restlessness, which is often relieved by blue-pill, alkalies, and bromide of potassium.

7. *Orchitis,* sometimes acute and sometimes chronic, is another result of lithæmia or gout. The chronic form is often attended by hydrocele and sometimes leads to the formation of indurated masses in the testicle or epididymis, which are mistaken for tubercle or cancer, but which disappear under time and treatment.

IX. *Abnormal Conditions of the Skin.*

There is good evidence that many disorders of the skin originate in derangements of the processes of oxydation or disintegration which go on in the liver.

1. Almost all observers are agreed that *Eczema, Lepra, Psoriasis,* and *Lichen* may arise from lithæmia. Many years ago the late Sir Henry Holland remarked that he had 'so often seen psoriasis prevailing in gouty families—sometimes alternating with acute attacks of that disease, sometimes suspended by them, sometimes seeming to prevent them in individuals thus disposed—that it is difficult not to assign the same morbid cause to these results.'[1] Our late distinguished President, Sir Thomas Watson, in his 'Practice of Medicine,' speaks of lepra and psoriasis as blood-diseases depending upon some poisons bred within the body.[2] Dr. Garrod also bears testimony to the frequent connection of eczema and psoriasis with gout; while Sir James Paget has pointed out that those patients in whom the local application of arnica to the skin is followed by erysipelas, with great pain, vesication, and desquamation, are always of gouty constitution.[3] My own experience fully bears out the correctness of these observations; but in many cases these cutaneous diseases appear to arise from the functional derangement of the liver which precedes or attends gout, and yet neither the patient nor any member of his family has ever suffered from this disease. On this point Dr. Tilbury Fox,

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[1] Medical Notes and Reflections.

[2] Lectures on the Principles and Practice of Medicine, 5th edit. 1871, vol. ii. p. 1023.

[3] Brit. Med. Journ. 1875, i. 633.

blood-changes of which the liver is the principal seat. Asthma, like gout, is an hereditary disease; it is common among persons springing from a gouty stock; it is not unfrequently associated with gout, gall-stones, or other hepatic derangements in the same individual; and attacks of asthma have been known to alternate periodically with attacks of gout. Moreover, an asthmatic paroxysm, like an attack of gout, of vertigo, or of sleeplessness, is often excited by a fit of indigestion and by the use of particular articles of diet. Our late colleague Dr. Hyde Salter, who did so much to throw light upon the pathology of asthma, was of opinion that the asthmatic paroxysm in the cases now referred to was produced by 'the actual presence in the vessels of the lungs of the materials taken up from the stomach and intestines;'[1] but it seems to me that the *materies morbi* is far more likely to be a product of hepatic derangement consequent on the unwholesome ingesta, as in the analogous attacks of gout, vertigo, etc.

VIII. *Derangements of the Genito-Urinary Organs.*

The remarks which I have already made render it unnecessary for me to insist further on the tendency of functional derangement of the liver to produce urinary symptoms. I will merely repeat that hepatic disorder is a common cause of—

1. *Deposits of Lithic Acid and Lithates in the Urine* (p. 565).
2. *Renal Calculi* (p. 568).
3. *Diseases of the Kidneys and Albuminuria* (p. 572).
4. *Cystitis* is occasionally excited by the lithic acid diathesis. It is often preceded by an excess of lithic acid in the urine, by a disappearance of an eczematous eruption, or by an attack of dyspepsia. The attack is often sudden in its invasion and rapid in its subsidence.
5. *Urethritis.*—Not only may lithæmia modify or protract an ordinary gonorrhœa, but it is sometimes the primary cause of acute urethritis. On this point I cannot do better than quote the words of Sir James Paget:—'Acute inflammation of the mucous membrane of the urethra, attended with the usual signs of gonorrhœa—purulent discharge, scalding, frequent micturition, and painful erections—occurred as a direct consequence of gout. These cases were certain; they occurred where there had been no infection and they were not themselves infectious.'[2]

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[1] On Asthma, 1860, pp. 46, 117.

[2] Brit. Med. Journ. 1875, i. 701.

have had under my care a boy, aged 9, with *urticaria tuberosa* and *purpura urticans*, complicated with hæmorrhages from the bowels, kidneys, and urinary passages, and with the discharge of much lithic acid in the urine, which there was good reason for suspecting to be due in the first instance to functional derangement of the liver.

3. *Boils and Carbuncles* are occasionally observed in connection with jaundice and are also excited by the presence in the blood of urea and other effete products. In the connection also between phlegmonous or carbuncular inflammation and diabetes, it is possible again to trace the influence of a disordered liver in the production of skin-diseases.

4. *Pigment-spots* of various sorts on the face, hands, and other parts of the body are not uncommon in functional derangements of the liver. They are sometimes designated 'liver-spots' by non-professional persons, who, perhaps, attach too much importance to them as indicative of hepatic derangement. They may, as Dr. Laycock observes, be induced by imperfect oxydation, or excessive production of carbon, in derangements of the liver,[1] but they may also arise in other ways.

5. *Xanthelasma or Vitiligoidea*, which consists in a fatty degeneration of the subcutaneous or submucous tissue analogous to atheroma, is a remarkable affection of the skin, often associated with liver-derangement. (See Lecture VII., p. 247; and Lecture IX. p. 318).

6. *Pruritus* is a troublesome symptom which often results from hepatic derangement. It is known to be a frequent accompaniment of jaundice; but it is not due to the presence of bile in the blood, for in many cases of jaundice it is absent, and I have repeatedly known it precede the appearance of jaundice by several weeks, or cease while the jaundice persisted. Moreover, I have frequently known itchiness of the skin a source of extreme distress to patients with hepatic derangement unaccompanied by jaundice. It may attack various parts of the body in succession, or it may be universal. It is unattended by any eruption. It is always worse in heated rooms and after stimulating food, and it is greatly aggravated by scratching; not unfrequently it appears with the advent of winter. This symptom is not uncommon in gouty people[2] and in subjects of the lithic acid diathesis, and it is often removed by attention to diet and a few doses of blue pill and alkalies.

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[1] Fox, op. cit. p. 404.

[2] Scudamore, op. cit. p. 103.

As Dr. Bence Jones has observed, 'itching, nettle-rash, eczema, and herpes are the outbursts of an over-acid state.'[1]

C. CAUSES OF FUNCTIONAL DERANGEMENTS OF THE LIVER.

The remarks which I shall make under this head will be restricted to abnormal disintegration in the liver. The causes of diabetes and of certain other functional derangements of the liver have been already referred to. The disorder of the liver which induces lithæmia may be primary, or secondary to other morbid states of the body. It is with the former that we are now chiefly concerned; but the main causes of secondary derangement of the liver may be briefly referred to. They are as follows:—

1. *All structural diseases of the liver* derange more or less the functions of the organ. These derangements are usually judged of solely by the characters of the alvine evacuations, and the far more important functions of sanguification and depuration of the blood performed by the liver are lost sight of. But it is well to remember that in structural diseases of the liver these functions may be seriously deranged, without any obvious change in the characters of the stools. In all structural diseases of the liver unattended by fever and involving a considerable destruction of the glandular tissue, there is a tendency to a diminished excretion of urea and an increase of lithates in the urine, and before long the patient becomes anæmic. At last, symptoms of blood-poisoning may supervene, although there may be no jaundice and plenty of bile in the motions. These results are well seen in acute atrophy of the liver; but are also notable, though in a less degree, in abscess, cirrhosis, cancer, etc.

2. *Disorders of the Gastric and Intestinal Digestion* often lead to secondary derangement of the liver. For example, the liver may become deranged as the result of gastric dyspepsia, or of protracted constipation from atony of the bowels or from deficient intestinal secretion; and sometimes it may be difficult to say whether the hepatic derangement is primary or secondary.

3. *Diseases of the Heart and Lungs*, by obstructing the circulation and impeding oxydation, are a common cause of functional and ultimately indeed of structural disease of the liver.

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[1] Lectures on Pathology and Therapeutics, 1867, p. 84.

It is unnecessary for me here to insist on the frequency with which the symptoms of valvular disease of the heart are aggravated by those of functional derangement of the liver, and on the necessity of attending to these in the treatment of the primary disease.

4. *Pyrexia.*—In all diseases attended by pyrexia, whether arising from some general cause, such as a specific poison, or from a local inflammation, there is more or less functional derangement of the liver. The liver, indeed, plays a prominent part in the pathology of the febrile process. It is one of the few parts of the body which do not waste during the fever. On the contrary, it becomes enlarged and congested, while its gland-cells are swollen out with minute albuminous granules; and it is well known that these changes are attended by an increased disintegration of albuminous matter and an increased production of urea and less oxydised products. On the cessation of the febrile process the liver resumes its normal functions; but now and then it happens that after a severe attack of fever these functions are permanently impaired. I have repeatedly known a permanent tendency to hepatic derangement induced by a severe attack of typhus, enteric, malarious, or scarlet fever, in persons who had exhibited no such tendency previously.

Functional derangements of the liver, when primary, may be due to a variety of causes. Of these the principal are—

1. *Errors in Diet.*—There can be no doubt that the present system of living and especially the consumption of even what are regarded as average quantities of rich food and stimulating drinks contribute largely to derange the liver. It will be generally admitted, nor would it be difficult to prove, that most persons are in the habit of eating a quantity of food far greater than suffices to maintain the nutrition of the body. Much of this excess is fortunately never assimilated and is got rid of in the fæces: but very often much more is taken into the blood than can be converted into tissue or pass through the ordinary processes of oxydation preparatory to elimination. The result is that the excess of food is thrown out in an imperfectly oxydised form by the kidneys, lungs, etc., or accumulates in the system; while more work is thrown upon the liver than it can readily perform, and functional derangement of the organ ultimately ensues. With regard to individual elements of food, speaking generally, it may be said that the liver is most apt to

be deranged by saccharine and fatty substances. The derangement of the liver which leads to lithæmia or gout is more likely to be induced by even small quantities of these substances than by a moderate excess of purely nitrogenous food, such as meat. Cooked articles of diet containing a large proportion of both sugar and fatty matter are in many persons certain to derange the liver. The excess of carbon in these substances must either be deposited as fat or must take away the oxygen, so as to leave little free to act on the nitrogenous matter passing out from the tissues or derived from the food; and hence, as Dr. Bence Jones has observed in speaking of gout, 'with carbonaceous diet in excess, the whole of the uric acid from the tissues might pass off through the blood unoxydised.'[1] There are also constitutional peculiarities with regard to many articles of food, which always derange the liver in certain individuals, though they are comparatively harmless in others.

But of all ingesta the various alcoholic drinks are most apt to derange the liver. They do so in two ways. *a*. They may cause persistent congestion of the liver. Even small quantities of alcohol in healthy persons produce a temporary hepatic congestion: but if alcohol be taken in excess, or too frequently, the congestion of the liver becomes permanent and the functions of the organ are deranged. Like results may ensue from comparatively small quantities in certain persons, who may be said to have a constitutional intolerance of alcohol. Of course, if the congestion be long maintained, structural disease may follow. *b*. But wines and other alcoholic drinks often cause derangement of the liver, which a corresponding quantity of pure alcohol would not produce, and which, in fact, cannot be accounted for by any one ingredient of the offending liquid—neither by the free acid, the ether, the salts, gum, sugar, nor extractive matter. This general rule, however, I believe holds good, that the injurious effect of alcoholic beverages upon the liver increases in a direct ratio with the amount of sugar plus alcohol which they contain. It would seem, indeed, that a mixture of alcohol and sugar produces injurious results which would not be caused by the admixture of a much larger quantity of sugar, or of alcohol alone, with the food. In accordance with this view, the alcoholic drinks which are found from experience to be most apt to disagree with the liver are malt liquors of all sorts, but especially porter and the stronger forms of mild ale, port wine, madeira, tokay, malaga, sweet champagne, dark sherries, liqueurs, and brandy; whilst those which are least likely to derange the functions of the organs are claret, hock, moselle, dry sherry, and gin or whisky largely diluted.

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[1] Op. cit. p. 142.

Derangement of the liver from excessive eating or from other errors in diet usually first shows itself in middle life—from thirty-five to forty-five. Young people, who take much exercise and whose bodies are still undergoing development, require more food and can with impunity eat more than they require. But by the age of forty the body is fully developed and most persons take less exercise than before, while at the same time they often indulge more freely at table. At any age errors in diet will be all the more likely to tell upon the liver, if there be any constitutional weakness in the functional power of the organ.

2. *A Deficient Supply of Oxygen.*—Insufficient muscular exercise in the open air may derange the functions of the liver. It is well known that sedentary habits and confinement in badly ventilated rooms tend to induce derangements of the liver. It is also a common observation that persons who have eaten and drunk too freely have not suffered from their livers so long as they have led an active life in the open air; but that, as soon as from change of occupation, or other causes, they take to sedentary habits, without any corresponding change in diet, derangement of the liver ensues. Again, every sportsman who has suffered from hepatic derangement knows the effect of a single day's hunting or shooting in clearing his complexion and relieving his symptoms. A want of regular exercise in the open air leads to derangement of the liver in two ways; viz., *a*, by diminishing the elimination of free acid and causing a deficient supply of oxygen to the system, as the result of which the oxydising processes which go on in the liver and elsewhere are imperfectly performed, and there is a tendency to the accumulation in the system of fat and of the imperfectly oxydised products of disintegrated albumen. Oxygen is, so to speak, the antidote necessary for the destruction of a *materies morbi* (lithic acid, etc.) produced by imperfectly oxydised albumen. *b*. By retarding the circulation of blood through the liver. Since the time of Haller,[1] physiologists have recognised the influence of the respiratory movements in promoting the circulation of blood

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[1] 'Vires quæ sanguinis per hepar motum accelerant.'—Haller's Physiologia, 1764, tom. vi. p. 601.

through the liver; but, upwards of thirty years ago, Mr. Alexander Shaw, in a paper which has attracted too little notice,[1] showed more clearly than ever before that the circulation of blood through the liver was greatly influenced by the alternate expansion and contraction of the thorax during respiration. Mr. Shaw called attention to the fact that the portal vein, without any provision for increasing its power, or any assistance beyond that *vis a tergo* which belongs to the veins generally, and being even destitute of valves to protect it from regurgitation of blood like the veins in other parts of the body, has to perform the duty usually fulfilled by an artery, which, besides receiving an impulse from the heart, is aided in distributing its blood by the contractility and elasticity of its coats. He suggested that this weak power by which the portal vein propelled its blood was compensated for by a suction-force communicated to the current of blood by the actions of respiration: the deeper the inspiration, the greater the force with which the blood rushes by the large veins to the right auricle. These reasonings have been confirmed by certain experiments of M. Bernard, who has found that when an incision is made into a lobe of the liver in a living animal the blood may be seen to jet from the mouth of the hepatic veins during the movements of expiration, but to return sucking in air with it at each deep inspiration, so that the animal soon dies from the passage of air into the heart.[2] In persons, then, who lead a sedentary life this auxiliary force for promoting the circulation of blood through the liver is diminished, blood stagnates in the gland, and the functions of the organ are deranged, these results being all the more likely to arise if the liver be at the same time overstimulated by errors in diet.

3. *A high temperature* favours certain functional derangements of the liver, and particularly those relating to sanguification and disintegration of albumen. Functional derangements, as well as congestion and inflammation of the liver, are more apt to occur in tropical than in temperate climates, and in our own country the liver more often becomes disordered in summer and autumn than in winter. The diet which is suitable in a cold or temperate climate produces in the tropics hepatic derangement. These results of a heated atmosphere are, no doubt, due in part to the rarefaction of the air and a correspond-

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[1] Medical Gazette, July 15 and September 30, 1842.

[2] London Medical Record, October 15, 1873, p. 647.

ing diminution in the supply of oxygen to the system; the hotter the air, the less will be the amount of oxygen in a given volume inhaled by the lungs. But this is, perhaps, not the sole, if the chief, explanation. Experiment has shown that one of the effects of a high temperature upon the lower animals is to produce a degeneration of the parenchyma of the liver, its secreting cells becoming filled with minute granules and presenting appearances similar to those found after death from febrile diseases.[1] It is possible, then, that some of the functional derangements of the liver from which persons suffer in tropical climates may be owing to similar degenerations, not necessarily permanent, of the secreting cells.

4. *Nervous Influences.*—Many facts show the great influence of the nervous system upon the secreting organs. Sudden fear, or other severe mental emotion, has been known to arrest the secretion of milk and saliva, and we have already seen how injuries and diseases of nerve-tissue may produce diabetes by deranging the glycogenous function of the liver. But many other ailments of the liver besides diabetes have a nervous origin. Prolonged mental anxiety, worry, and incessant mental exertion not only interfere with the proper secretion of bile, but too often derange the processes of sanguification and blood-change, in which the liver is so deeply concerned, and induce lithæmia with many of the symptoms already described. Gravel and gout are acknowledged to be the frequent lot of those who live more by nerve- than by muscle-work. Such results are all the more likely to ensue if the diet has been such as favours hepatic derangement—if, for example, to drown grief, the patient has indulged in stimulants—and the habits have been sedentary. There is also good evidence that nervous agencies may not only cause functional derangement, but even structural disease, of the liver. Acute atrophy, in which the secreting cells are rapidly disintegrated and the functions of the organ arrested, appears in many instances to have a purely nervous origin; very often the first symptoms of the disease have occurred immediately after a severe fright, or an outburst of passion, in a person previously healthy. An impression made upon the brain appears to be reflected to the liver and to derange its nutrition. Many observations have satisfied me that the extrusion of gall-stones from the gall-bladder, as well as their formation, may be traced to nervous

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[1] See Pathological Transactions, 1873, vol. xxiv. p. 266.

agency. Dr. Budd has also observed that mental anxiety or trouble has 'great influence in the production of gall-stones;'[1] and I have repeatedly known attacks of biliary colic from gall-stones excited by some sudden emotion. Lastly, even cancer of the liver appears sometimes to result from the functional derangement induced in the first instance by mental trouble. I have been surprised at the frequency with which patients suffering from primary cancer of the liver have traced the commencement of their ill-health to indigestion following protracted grief or anxiety. The cases have been far too numerous to be accounted for on the supposition that the mental distress and the cancer have been mere coincidences. A similar observation has, I believe, been made by Sir Robert Christison and by other eminent authorities.

5. *Constitutional Peculiarities.*—In considering the causes of functional derangement of the liver it must not be forgotten that there are constitutional peculiarities—inherited or acquired—in virtue of which the liver is deranged from causes which under ordinary circumstances would be harmless. Most persons, as Dr. Budd observes, have more liver, just as they have more lung, than is absolutely necessary.[2] A portion of their liver may be destroyed by disease, or become less active, without any derangement of the general health. In others, the liver seems only just capable of performing its functions under the most favourable conditions, and it at once breaks down under adverse circumstances of diet, habits, or climate. This innate weakness of the liver is often inherited. The person is born with a tendency to biliary derangements. Gout and diabetes, which we have found to originate in hepatic derangement, are hereditary diseases; and the liver is always very readily disordered in persons who inherit a tendency to gout. This constitutional tendency to hepatic derangement is too often lost sight of by patients, and perhaps sometimes by their medical advisers. The habitual use of alcohol is often recommended for various ailments by the medical attendant, without due regard to the tendency of the individual to hepatic derangement, and thus serious consequences may ultimately arise from alcohol taken with a medicinal object. Again, a patient often argues that his liver-troubles cannot be due to what he eats or drinks, because he is most careful as compared with friends who indulge largely and suffer nothing, forgetting the adage: 'One man's

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[1] Diseases of the Liver, 3rd edit. 1857. p. 369.

[2] Op. cit. p. 55.

food is another man's poison.' One man, for instance, may drink a bottle of wine and be none the worse, whereas another has his liver deranged by a single glass.

6. *Poisons* of various sorts may derange the liver, the persistence of the derangement depending upon the length of exposure to the cause. One of the first effects of the poisons of the various specific fevers is upon the liver. Again, from protracted exposure to malaria, the liver often becomes deranged, anæmia and lithæmia being the results. While some cases of acute atrophy of the liver have a nervous origin, there is good evidence that others are caused by a poison taken into the body from without, or sometimes, as suggested by Dr. Budd, 'engendered in the body by faulty digestion and assimilation.' Phosphorus also, in sufficient doses, is known to derange the functions of the liver; and one result of phosphorus-poisoning is the production of symptoms and structural changes in the liver closely resembling those of acute atrophy.[1] Various substances also taken as food, or by mistake along with food, may induce functional disturbance of the liver, the poisonous effect being often determined by some constitutional peculiarity of the individual.

These causes of functional derangement of the liver will, of course, act more injuriously if the functions of the liver be already disordered by structural disease, by disease of the heart or lungs, or by derangements of the stomach and bowels.

D.—TREATMENT OF FUNCTIONAL DERANGEMENTS OF THE LIVER.

The time at my disposal only permits me to give a brief sketch of the general principles on which functional derangements of the liver ought to be treated; and my remarks will be for the most part restricted to the derangements resulting from abnormal disintegration and abnormal elimination.

1. *Diet.*—In functional derangements of the liver, much more permanent benefit is to be expected from careful regulation of the ingesta than from physic. It must not be forgotten that what may ultimately destroy the body too often enters by

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[1] Lead is known to cause an accumulation of lithic acid in the system, but apparently by impeding its excretion by the kidneys, rather than by increasing its formation in the liver. (See Garrod, op. cit. p. 292.) Bence Jones, on the other hand, maintains that the accumulation of lithates in the system produced by lead is to be ascribed to diminished oxydation. (Lectures on Pathology and Therapeutics, p. 289.

the same portal as that which is intended to nourish and maintain it, and that for the maintenance of health it is necessary for most persons to put a curb upon their appetites. To use the words of the late Sir Benjamin Brodie, 'We are all anxious to obtain rank, reputation, and wealth; but that for which we have most reason to be anxious, not only for our own sake, but also for that of others, is such a state of our bodily functions as will enable us to make use of our higher faculties, and promote in us happy and contented feelings. . . . . The agricultural labourer who has enough of wholesome food and warm clothing for himself and his family, and who has the advantage of living in the open air, has more actual enjoyment of life than the inheritor of wealth living in a splendid mansion, who has too much of lithic acid in his blood.'[1] It is also well to remember that the hepatic derangement resulting in lithæmia may exist for years without any other symptom than the frequent deposit of lithates or lithic acid in the urine, and is then curable by attention to diet alone; but that, if neglected, it may ultimately develope gout, structural disease of the liver or kidneys, or some other serious malady. Habitual lithæmia ought, therefore, to be always counteracted, and, from what has been already stated, it seems clear that the foods mainly to be avoided are saccharine and oleaginous articles, and especially cooked dishes containing both of these substances. Patients with lithæmia ought always to avoid made-up or highly seasoned dishes. In severe cases, potatoes, rice, sago, and fruits may have to be given up, and even bread must be taken in moderation. It will also always be well to ascertain if the lithæmia be due to any of those idiosyncrasies in virtue of which particular articles of diet are apt to derange the liver. In most cases of lithæmia, a diet consisting chiefly of stale bread, plainly cooked mutton, white fish, poultry, game, eggs, a moderate amount of vegetables, and weak tea, cocoa, or coffee answers best; while in others the patient enjoys best health on a diet composed of milk, farinacea, vegetables, eggs, and occasionally fish. The quantity, as well as the quality, of the food must be attended to. Habitual lithæmia often results from the patient taking more food than can be converted into tissue or disintegrated in the liver. As Dr. Bence Jones has observed with regard to gout, so in habitual lithæmia, 'a minimum of albuminous food should be taken, in order to produce the least uric acid; and a minimum of carbonaceous food, in order to allow the uric acid to be oxydised as much as possible.'[1] In obstinate cases the patient may be advised to take his principal meal in the morning, when the digestive powers are strongest.

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[1] Psychological Inquiries, 2nd edit. 1855, p. 76.

Still greater caution is necessary in all forms of lithæmia as to alcoholic stimulants. Malt liquors, port wine, champagne, and many other wines ought to be strictly prohibited. Claret, or a small quantity of spirit largely diluted, as a rule, answers best; but even these should be taken sparingly, and many patients do best with no stimulants at all. This is not the occasion to discuss whether alcohol is necessary for the nutrition of the body in persons subjected to much mental or bodily toil, or whether, taking the masses in all walks of life, the standard of health would be better maintained by teetotal habits or by a moderate use of alcohol. What I desire now to insist upon is, that alcoholic drinks in quantities usually regarded as compatible with, if not conducive to, health, and far short of what are necessary to affect the brain, in many persons undermine the foundations of health by deranging the liver; and that to some individuals even very small quantities are injurious. In persons who have been indulging largely, the risk of a sudden withdrawal of stimulants is less, I believe, than is commonly imagined. Unless there be evidence of a very weak heart, which itself may be the result of alcohol, the only unpleasant effects of sudden and complete abstinence, in my experience, have been sensations of sinking at the epigastrium and craving for alcohol, which a repetition of the stimulus has only temporarily relieved and has rendered more persistent.

2. *A Free Supply of Oxygen.*—Next to careful regulation of diet, this is the most important object to be aimed at in the treatment of functional derangement of the liver, and especially in that which induces imperfect disintegration of albumen. An excess of fresh air, indeed, will often counteract the bad effects of too large a quantity of food. Although recent observations, more especially those of Parkes, have shown that the common impression that muscular exercise materially increases the elimination of nitrogen from the body is erroneous, there can be no doubt that exercise in the open air quickens the circulation of blood through the liver in the manner already explained and promotes oxydation, and that, by thus preventing the accumulation in the system of the imperfectly oxydised products of albumen, it

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[1] Op. cit. p. 142.

operates beneficially in the treatment of functional derangement of the liver attended by lithæmia. The observations of Beneke and other authorities have shown that *sea-air* is an oxydising agent of great power, and that nitrogenous and sulphur-holding tissues more rapidly disintegrate under its influence.[1] Accordingly we find that many patients with hepatic derangement and lithæmia derive immense benefit from residence at the seaside and sea-bathing, although unfortunately the good effects of sea-air are sometimes more than counterbalanced by unhealthy lodgings or improper and badly cooked food.

3. *Diluents.*—The free use of diluents, such as soda and seltzer waters, is also useful, as they help to eliminate from the system the morbid products of disintegration. Many patients also with lithæmia derive great benefit from drinking half a pint of cold water, or of some alkaline water, while dressing in the morning and before going to bed.

4. *Baths.*—In all cases of lithæmia and gout the action of the skin ought to be maintained by frequent bathing or ablutions of the entire body with tepid water and soap. Cold baths are often objectionable from inducing muscular or gouty pains, or internal congestions.

5. *Aperients; Cholagogues.*—In a large number of cases of functional derangement of the liver great advantage is derived from the frequent use of aperient medicines, whether there be a tendency to constipation or not. Aperients bring away not merely bile, but the products of disintegration contained in the fluid circulating between the liver and bowel prior to their further elaboration and elimination by the lungs and kidneys. Saline aperients, from the promptness of their action and the large quantity of watery exhalation from the bowel which they induce, are among the best for the purpose now mentioned. Recourse is usually had to the sulphate of magnesia (Epsom salt), the sulphate of soda (Glauber salt), the tartrate of potash and soda (Rochelle salt), or the phosphate of soda,[2] or to various combinations of these salts with chloride of sodium, carbonate of soda, and other alkaline salts, such as are found in the mineral waters of Carlsbad, Friedrichshall, Püllna, Harrogate, or Cheltenham, or in the recently discovered Hungarian spring, Hunjadi

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[1] Parkes on Urine, 1860, pp. 115, 129.

[2] Professor Rutherford informs me that he has found the sulphate of soda and the phosphate of soda to be powerful excitants of the biliary secretion in dogs, but the sulphate of magnesia to have no effect at all.

János. Daily experience shows the great benefit derived by patients with lithæmia from a course of one or other of these mineral springs, or from some artificial imitation of them, all of which are best taken with warm water and in the morning fasting. All of these salts have little or no affinity for animal textures, so that they excite few changes in them; they cause very little irritation of the mucous membrane of the bowel, and do not excite peristalsis, so that they purge without producing griping or pain. They act apparently by preventing the reabsorption of the fluid which is constantly being exhaled from the blood-vessels into the bowel.

There are certain other aperients which have long enjoyed a great reputation for promoting the secretion and discharge of bile, and otherwise acting beneficially in derangements of the liver, and which have accordingly been designated *Cholagogues.* Among these remedies *mercury* and its preparations hold a pre-eminent place. At the present day mercury has lost much of its former reputation as a cholagogue and alterative, and there is much difference of opinion as to its power over the liver. The practical physician gives a dose of calomel, finds the quantity of bile in the motions greatly increased, and his patient's state much improved; and he argues that the liver has been stimulated by the mercury to an increased secretion of bile, and that to this cause his patient's improvement must be ascribed. The physiologist, on the other hand, ties the common bile-duct in one of the lower animals, produces a fistulous opening into the gall-bladder, and then finds that calomel has no effect on, if it do not diminish, the amount of bile that drains away through the fistula. It may interest some who are present if I refer briefly to the principal of these experiments.

Kölliker and Müller, in 1855, tried the effects of calomel upon the secretion of bile in a dog with a biliary fistula. The results were somewhat contradictory. Once the bile seemed to be increased, and twice it seemed to be diminished, by the administration of calomel.[1]

Of four experiments made in 1858 on a dog with a biliary fistula, Dr. George Scott found that in all the administration of large doses of calomel was followed by a diminution of fluid bile and of bile-solids.[2]

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[1] Wurzburg Verhandlungen, Bd. v. 1855, s. 231.

[2] Beale's Archives of Medicine, 1858, vol. i. p. 209.

In the same year (1858), Dr. Mosler made similar experiments upon two dogs with biliary fistulæ. The administration of calomel was not followed by any increase of bile, nor could mercury be detected in the biliary secretion.[1]

Ten years later (1868), a committee of the British Medical Association, with Professor Hughes Bennett of Edinburgh as chairman, made a number of similar experiments on dogs, and came to the conclusion that 'mercury did not increase the flow of bile, but rather diminished it.'[2]

Next in order (1873) come the experiments of Dr. Röhrig of Kreuznach, made in the Pathological Institute of Vienna. He found that, although large doses of calomel did seem to increase somewhat the secretion of bile, its power to do so was inferior to that of croton oil, colocynth, jalap, aloes, rhubarb, senna, and sulphate of magnesia, the cholagogue power of these drugs diminishing very much in the order in which they have now been enumerated, and calomel standing at the bottom of the scale.[3]

The most recent experiments are those of Professor Rutherford and M. Vignal on four different dogs during fasting. In three the secretion of bile was diminished, and in one it was ascertained that not only the total quantity, but the percentage of solids, was reduced. In the fourth case the quantity of bile was increased, but there were reasons for believing that the increase was not due to the calomel.[4]

These results of experiments upon the lower animals have added greatly to the discredit previously thrown upon mercury by its failure, when brought to the test of accurate clinical observation, to absorb plastic lymph in most forms of inflammation; and some eminent physicians are even of opinion that mercury and its preparations ought to be erased from our Pharmacopœia.[5] On the other hand, it has been fairly objected that the results of experiments with mercury upon dogs do not warrant conclusions as to its effects upon man; and even granting that in man mercury does not increase the quantity of bile secreted by the liver in health, it does not follow that in disease there may not be some condition adverse to the formation of bile, which mercury may have the power of removing. Much, however, of the difference of opinion between the physiologist and the practical physician may be reconciled by keeping in mind the osmotic circulation, to which I referred in my first lecture (p. 550), as constantly going on between the intestinal contents and the blood. A large part of the bile secreted by the liver and thrown into the bowel is constantly being re-absorbed, to reach the liver again; and accordingly, when the common bile-duct is tied and a fistulous opening into the gall-bladder established, the quantity of bile which escapes from the fistulous opening immediately after the operation is much greater than at any time subsequently (Schiff). Mercury and allied purgatives produce bilious stools by irritating the upper part of the bowel and sweeping on the bile before there is time for its re-absorption. The fact of mercury standing at the bottom of the scale of cholagogues in Röhrig's experiments is accounted for by its surpassing other cholagogues in this property; for of course the larger the quantity of bile that is swept down the bowel, the less is re-absorbed and the less escapes from a biliary fistula. That mercury does act especially upon the duodenum, is proved not merely by the large flow of bile which follows its action, but by the fact discovered by Radziejewski, that leucin and tyrosin, which are products of pancreatic digestion, under ordinary circumstances decomposed in the bowel, appear in the fæces after the administration of mercurials. It would appear, then, that mercury, by increasing the elimination of bile and lessening the amount of bile and of other products of disintegrated albumen circulating with it in the portal blood, is after all a true cholagogue, relieving a loaded liver far more effectually than if it acted merely by stimulating the liver to increased secretion, as was formerly believed, and as some authorities still maintain; for in this case it might be expected to increase, instead of diminish, hepatic congestion.[1] It is not impossible, also, that the irritation of the duodenum by calomel and other purgatives may be reflected to the gall-bladder, and cause it to contract and discharge its contents, and thus account in part for the increased quantity of bile in the stools.

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[1] Virchow's Archiv, 1858, Bd. xiii. s. 29.

[2] British Medical Journal, 1868, vol. ii. pp. 78, 176, 191.

[3] Stricker's Jahrbuch, 1873, part 2.

[4] British Medical Journal, Nov. 13, 1875.

[5] See Bennett, British Medical Journal, 1868, vol. ii. p. 176.

There are likewise, I believe, grounds for believing that, apart from its increasing the discharge of bile from the bowel, mercury exerts a beneficial action in many functional derangements of the liver, in whatever way this is to be explained. Patients of the greatest intelligence suffering from hepatic disorders constantly declare, that they derive benefit from occasional or repeated doses of mercurials which no other medicine or treatment confers; and the scepticism of the most doubting physician would, I believe, be removed, should he unfortunately find it necessary to test the truth of their statements in his own person. It is not impossible that the good effects of mercury on the liver, and in some forms of inflammation, may be due to its property of promoting disintegration. Mercury appears to have the power of rendering effused fibrin less cohesive, and so more easily removed by absorption, than it otherwise would be.[1] Modern physicians of high standing, and little likely to be accused of credulity as to the beneficial action of drugs, have thought that mercury is useful in croup, by causing a degradation and disintegration of the plastic membrane, and this view would explain why it is that mercury is always injurious in persons of scrofulous constitution. If this view be correct, it seems also not improbable that mercury, which from experiments[2] we know to reach the liver, may under certain circumstances act beneficially by promoting, or in some way influencing, the disintegration of albumen. The remarkable effect of mercury on constitutional syphilis perhaps admits of a similar explanation. The effect of mercury on the elimination of nitrogen by the kidneys has still to be investigated. But in whatever way it is to be explained, the clinical proofs of the efficacy of mercury in certain derangements of the liver are to my mind overwhelming. I say so the more advisedly, because I was taught to regard mercury as a remedy worse than useless, not only in hepatic diseases but in syphilis; it cannot, therefore, be said that the convictions forced upon me by experience are the result of preconceived opinions.

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[1] This view as to the action of mercury upon the liver has been taught by me in my lectures for many years, and was enunciated in the first edition of my Clinical Lectures on Diseases of the Liver, published in 1868, pp. 126, 309, 404.

Podophyllin is a remedy which seems to act in a very similar manner to mercury. In small doses it has been shown by Professor Rutherford to increase the secretion of bile, but in decidedly purgative doses, which are usually necessary to relieve the liver, it diminishes the biliary secretion. Dr. Anstie's experiments with podophyllin on dogs and cats show that it has a special affinity for the small intestines, and especially for the duodenum. So far as my experience goes, it is less certain in its action, and even in moderate doses more likely to cause griping and mucous stools, than the preparations of mercury. It is a good substitute, however, for mercury, when from any cause this is contraindicated.

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[1] Bence Jones, op. cit. p. 283.

[2] Authenrieth and Zeller found mercury in the bile of animals treated with mercurial frictions. (Budd, op. cit. p. 57.)

Colocynth, *Aloes*, *Rhubarb*, *Jalap*, and *Senna* are also useful aperients in functional derangements of the liver resulting in lithæmia, constipation, or deficient excretion of bile. Röhrig's experiments on dogs seem to show that they actually increase the amount of bile secreted by the liver: while from those of Professor Rutherford and M. Vignal it may be inferred that, with the exception of senna, they are cholagogues of considerable power.

Ipecacuanha, which I have already referred to (p. 136), as a powerful remedy in disease of the liver, I have also often found of great service in functional disorder of the gland, and from Professor Rutherford's experiments on dogs it would appear to be one of the most powerful known cholagogues.

Colchicum has also been found by experiment to be a cholagogue in dogs, and in man it is a useful adjunct to other aperients in cases of liver-derangement with lithæmia. According to Dr. Garrod, it 'may often be given with advantage to gouty subjects as a cholagogue in lieu of the preparations of mercury,'[1] which, I may add, are often contraindicated in chronic gout with renal disease.

'*Iridin*,' obtained from the root of the Iris versicolor, '*Euonymin*,' prepared from the bark of the Euonymus atropurpureus, and '*Sanguinarin*,' obtained from the Sanguinaria plant, have a considerable reputation in America as cholagogues and alteratives of the hepatic functions. From the experiments of Professor Rutherford also they appear to be powerful hepatic stimulants, so that they are well worthy of a careful trial in the human subject.[2]

With these remedies we may include *Taraxacum*, which has long been thought to exercise a specific action upon the liver, but which has been proved to be a very feeble hepatic stimulant, and probably acts mainly as a mild aperient. When there is a tendency to constipation it may be advantageously combined with either alkalies or mineral acids.

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[1] Op. cit. p. 410.

[2] Experiments on the Biliary Secretion of the Dog. (Journal of Anatomy and Physiology, vol. xi. part 1, 1876.)

6. *Alkalies.*—Next to aperients, alkalies are the most useful drugs in the treatment of functional derangements of the liver. In lithæmia and in many of the symptoms which spring from this morbid state, the greatest benefit is often derived from a course of alkalies—such as the alkaline salts of potash, soda, or lithia, or some of the alkaline mineral waters, such as those of Vals, Vichy, or Ems. The comparative worth of the different alkalies for neutralising acids varies considerably. One grain of carbonate of lithia or of carbonate of ammonia is nearly equal to a grain and a half of carbonate of soda or two grains of carbonate of potash. The beneficial effects of alkalies in derangements of the liver are not due to their neutralising acidity, or to any direct action upon lithic acid. It is, in fact, in the form of lithate of soda that lithic acid is met with in gouty persons. Alkalies seem to do good by combating the pathological state on which the formation of lithic acid depends. They are believed to promote oxydation, and thus to increase the disintegration of albumen. Dr. Bence Jones tells us that in the body as well as out of it alkalies furnish the most marked evidence of assisting in oxydising actions.[1] The experiments of Parkes with liquor potassæ seemed to show that it has the power of increasing the disintegration of the sulphur-holding materials of the body. The effect of its administration was to increase the amount of sulphuric acid and also of urea in the urine; although, with characteristic caution, Parkes adds that the increase of urea as the result of the potash was rendered probable, rather than proved, by his experiments.[2] From experiments on dogs with biliary fistulæ, Nasse was led to the conclusion that carbonate of soda taken with the food diminished greatly the secretion of bile;[3] and a similar result has been observed by Röhrig to follow the introduction of the same salt into the intestine or the veins; the diminution affected the solids as well as the water of the bile, and especially the biliary salts. Nasse also found that after taking two drachms of carbonate of soda the urine (human) was very rich in hippuric acid. The only inference at present to be drawn from these experiments is, that alkalies exert a powerful influence over the chemical changes going on in the liver. When alkalies are employed in lithæmia, it is well to suspend their use occasionally, as they are apt, when long continued, to

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[1] Op. cit. p. 280.

[2] On the Urine, 1860, p. 151.

[3] Archiv für Wissensch. Heilkunde, 1864, Bd. vi. p. 508.

derange the gastric digestion; but in cases where they are strongly indicated they are better tolerated than is usually thought. In the fifth volume of the Medico-Chirurgical Transactions Dr. Bostock has recorded the case of a young lady, who for months took carbonate of soda to the amount of 2½ oz. daily. The appetite and strength were much improved; and her blood, instead of being thin, coagulated firmly, the coagulum being strongly buffed and cupped.

7. *Chlorine*, *Iodine*, and *Bromine* are closely related in their chemical properties, and are believed to promote oxydation in the body by taking hydrogen from water and liberating oxygen. An aqueous solution of chlorine is of service in certain cases of lithæmia associated with general debility; and we know that the various salts of chlorine enter largely into the composition of the mineral waters which are most useful in hepatic derangements. Bromide of potassium will reduce certain enlargements of the liver and spleen, and may be given with advantage in cases of lithæmia associated with congestion of the liver and want of sleep. But among the remedies of this class the chloride of ammonium holds a pre-eminent place. It has obtained a great and well-deserved reputation in India and other tropical countries for the treatment of hepatic congestion; and I have found it of great service in the functional derangement of the liver attended by lithæmia. Given in scruple doses three times a day, it acts as a diaphoretic and diuretic and exercises a powerful influence in relieving the portal circulation. It is not oxydised, but passes out of the system unchanged in the urine. Professor Rutherford has found it to have no effect upon the bile-secretion of dogs; but according to Böcker's experiments,[1] it increases the nitrogenous solids of the urine; the mean daily increase of urea under its use he found to be not less than 74 grains—a quantity indicating a vast augmentation either of metamorphosis or of elimination, but from its beneficial effect on the liver, most probably of the former. Chloride of ammonium has also this advantage, that it may be combined with either alkalies or mineral acids. (See also Lecture IV., p. 136.)

8. *Mineral Acids* are employed by many physicians in the treatment of functional derangements of the liver. Nitric acid especially has long been thought to have the power of augmenting the flow of bile; but there is no good evidence of this,

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[1] Parkes, op. cit. p. 165.

either clinical or experimental. Professor Rutherford, indeed, informs me that nitro-muriatic acid has no effect upon the bile-secretion of dogs. According to my experience, the action of mineral acids upon the liver is much less direct than that of alkalies. In all morbid states of the liver attended by congestion and in most cases of lithæmia I have found that they either did no good, or that they aggravated the symptoms. They may, however, be of service when there is debility and want of tone; but the chief good which they effect is probably that of improving the gastric digestion. In some cases, both acids and alkalies may be given advantageously—the alkalies before, and the acids after, a meal.

9. *Tonics.*—Clinical experience shows that, notwithstanding the existence of debility and anæmia, tonic remedies are apt to disagree in many cases of functional derangement of the liver. This remark applies especially to the functional derangement resulting in lithæmia. I have repeatedly known patients in this state improve at once on substituting abstinence from alcohol, with aperients, blue pill, alkalies and careful regulation of diet, for quinine, iron, the mineral acids and stimulants; the strength, flesh, and colour returning under what at first sight might have appeared a lowering treatment. Different opinions have been expressed with regard to the utility of iron in chronic gout. According to Dr. Bence Jones, iron is one of the two most potent remedies we possess for indirectly promoting oxydation in gout as well as in other maladies;[1] whereas, in Dr. Garrod's opinion, the preparations of iron are very likely, when indiscriminately given to gouty subjects, to excite a paroxysm of the disease, and for the most part are contraindicated.[2] My experience coincides with that of Dr. Garrod; and in simple lithæmia I have constantly known iron to increase the tendency to deposits of lithates in the urine, constipate the bowels, and aggravate any symptoms from which the patient may have previously suffered. As a rule, also, I have found that patients with chronic gout or with lithæmia do not tolerate even small doses of quinine. From some careful experiments made by Dr. Ranke of Munich, quinine appears to have the power of diminishing the amount of lithic acid in the urine.[3] The experiments were made on three persons, and the results were uniform. The effect continued for about two days after a single dose of 20 gr.; and there was no evidence of any

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[1] Op. cit. pp. 143, 279.

[2] Op. cit. p. 453.

[3] Parkes, op. cit. p. 167.

increased excretion after the effect of the quinine had passed off, so that probably the quinine acted by lessening the formation of lithic acid in the liver, or by substituting some other substance for it. From these experiments it might be inferred that quinine and bark should be of essential service in chronic gout and in lithæmia, but this inference is opposed to clinical experience.[1]

When tonics are given in lithæmia, gentian, chiretta, cascarilla and serpentaria are preferable to quinine and other preparations of bark. The best preparations of iron are the reduced iron, the citrate of iron, or the tartrate of iron and potash; these preparations are sometimes advantageously combined with alkalies and saline aperients. In many cases, whether attended by flatulence or not, I have seen great relief follow the use of small doses of nux vomica or strychnia. In cases of lithæmia attended by great nervous prostration I have also seen advantage from the use of phosphorus in doses of one-thirtieth of a grain three times a day. The lithates have disappeared from the urine and all the symptoms have improved. In some of these cases the circumstances seemed to leave no doubt that the improvement was due to the phosphorus. Many patients also with lithæmia who cannot take iron derive benefit from arsenic, which has long been known to be of service in some of the complications of gout.

10. *Opium* and its preparations are contraindicated in most functional derangements of the liver, and particularly when there is evidence of lithæmia. Opium impedes elimination both by the bowels and kidneys and also appears to check the disintegrative processes which go on in the liver. It is generally believed to diminish the amount of bile secreted by the liver; and this view is favoured by the light-coloured stools which often follow its use. The result, however, of Röhrig's experiments on animals with biliary fistulæ was to show that opium increased the secretion of bile instead of diminishing it; so that probably the discharge of bile from the liver is only temporarily suspended by a similar influence on the coats of the bile-ducts to that which opium exerts on the coats of the bowel. Be this as it may, there can be no doubt that opium constipates the bowels, favours portal congestion, and checks the elimination, not only of bile, but of the products of disintegration which go on in the liver. The experiments of

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[1] Ranke's experiments have been repeated by Dr. Garrod, who found that quinine did not materially influence the excretion of lithic acid. Garrod, op. cit. p. 451.

Böcker[1] and clinical experience alike show that opium impedes the elimination of the nitrogenous solids of the urine, and that dangerous and even fatal consequences ensue from its employment in structural diseases of the kidneys. These considerations explain why in hepatic derangement attended by lithæmia opium is contraindicated for the relief of pain, sleeplessness, or other symptoms for which it is commonly prescribed.

On the other hand, it is not a little remarkable, as confirmatory of the supposed antagonism between gout and diabetes, to which I have already referred (p. 559), that in the functional derangement of the liver which exists in the latter disease opium is tolerated in large doses, and is often of signal service in checking the formation of sugar.[2] Its good effect is probably in great measure due to some influence on the vaso-motor nerves of the hepatic vessels, a reflex paralysis of which we have found to be one of the causes of diabetes.

In bringing these lectures to a close, I beg to tender to you, Mr. President, and to the Censors' Board, my thanks for the honour you have done me in appointing me to deliver them. I am very sensible of their manifold imperfections, and in apology can only plead a multiplicity of other engagements during the brief period allotted to their preparation. I am well satisfied, however, as to the great importance of the subject which I have brought before your notice, and that it is one worthy of more attention from the Fellows and Members of this College, and from the profession generally, than it has yet received. The day, I believe, will come when, with a more perfect knowledge than we now possess of the healthy functions and of the signs of functional derangement of the liver, we shall be enabled to prevent, or to arrest at their commencement, many of the most serious ailments to which mankind are liable, and thereby to add another chapter to the volume of Preventive Medicine.

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[1] Parkes, op. cit. p. 167.

[2] This is no new discovery. Sixty-three years ago, Sir Benjamin Brodie communicated to the Royal Medical and Chirurgical Society a case of diabetes treated with opium. Twenty-four grains of opium were taken in the day with the effect of reducing the urine from twenty-five to seven pints, but without any of the usual effects of opium. Medico-Chirurgical Transactions, vol. v. p. 236.

APPENDIX.

Page 75. Since Lecture III. passed through the press, the following case of hydatid of the liver, treated successfully by paracentesis, has come under my notice.

Case CLXXVII.—*Hydatid of Liver—Paracentesis—Recovery.*

William C——, aged 31, bootmaker, admitted into St. Thomas's Hosp. Dec. 5, 1876. Habits temperate; taken no stimulants for 14 years. Never out of England. As a rule digestion good, and no pain after food; but for a year or more liable at intervals of a week or month to attacks of flatulence and vomiting, usually in evening. A week ago first noticed a swelling in right hypochondrium, where for two or three weeks before there had been slight pain. Up to this time followed work. Tumour had enlarged slightly.

On admission there was a rounded, smooth, elastic, painless tumour in epigastric and right hypochondriac regions, causing ribs to bulge outwards, and throwing forwards belly of rectus, evidently connected with liver. Lower margin of tumour reached to umbilicus, and hepatic dulness in r. m. l., including tumour, measured 9 in.; girth of r. chest, 2 in. below nipple, 17¼ in.; of left, 15¾ in. Slight dull pain in hepatic region. All other organs healthy. Tongue coated; appetite bad; bowels regular. P. 84; temp. normal.

Dec. 13.—Paracentesis with fine trocar; 5 oz. of fluid drawn off, clear, sp. gr. 1009; much chlorides, but not a trace of albumen; no echinococci. Some hours after operation patient had a severe attack of abdominal pain, and temp. rose from 98° to 101·2°. Pain was relieved at once by subcutaneous injection of morphia gr. ⅓. Next day felt quite well, but temp. varied from 101·2° to 102·6°. On 14th, temp. normal and ap. good. After this had no bad symptom; and when patient left hospital on Dec. 23, tumour could not be felt, and girth on two sides of chest equal.

Page 134. Among the blood-poisons causing congestive enlargement of the liver, special attention ought to have been directed to syphilis. Both the liver and the spleen are not unfrequently found to be greatly enlarged in children who are the subjects of inherited syphilis, and that this enlargement is not waxy, and in all probability congestive, is shown by the rapidity and completeness with which it often disappears under treatment, or independently.[1] A similar enlargement is occasionally met with in adults in the secondary stage of syphilis, sometimes in conjunction with catarrhal jaundice. (See pp. 153, 157.)

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[1] See Brit. Med. Journ. 1877, i. 170.

Page 144. Case LII.—*Jan.* 9, 1877. Captain M—— continues in excellent health, and has applied for a command in the Pacific. Gained flesh; girth at umbilicus 30 in.; liver smaller; dulness in r. m. l. 5½ in.

Page 150. Case LVII.—*Jan.* 13, 1877. Mrs. R—— is much better; gained much flesh, and has been about and attending to business in her husband's (a baker) shop. Liver smaller. No periostitic pains, but a node on right ulna. Catamenia appeared a few days ago, the first time for two years.

Pages 142 and 283. Case CLXXVIII. is the one referred to in Lect. IV. of interstitial hepatitis resulting from a chill. I am indebted for the particulars to Dr. Wilson Fox, under whose care the patient was. In Case CLXXIX., where the hepatitis went on to cirrhotic contraction, the disease also probably originated in a chill. The case was also interesting from the early age of the patient (see pp. 282–300). Frerichs records the case of a boy aged 10, in whom cirrhotic contraction of the liver also appeared to originate from a chill in bathing.[1]

Case CLXXVIII.—*Interstitial Hepatitis resulting from a Chill.*

J. C——, 45, adm. into University College Hosp., with following history and symptoms.

For 4 years a cab-driver; before that had driven omnibus for 16 years. Married 27 years; 4 children living. Had lived all his life in London and been of temperate habits; had taken 1½ pint of beer, and quarter of a pint of claret, or a glass of port wine, daily; only very rarely taken a little rum. Strong corroborative evidence that this statement was reliable. No hereditary tendency to disease, except that father had been habitually intoxicated and had died in a fit at 55. Patient's previous health had been always good; never had syphilis, dyspepsia, nor morning sickness. Two months before admission had got wet through and remained thus on his cab for six or seven hours. On coming home felt chilly, and during night had pains in legs and ankles, which next morning were swollen. Next evening felt worse and had pains in shoulders, across chest, and in region of liver. He lost appetite, flesh, and strength, and vomited occasionally, and for a fortnight before admission he had vomited all solid food. A few days before admission had an attack of severe pain in epigastrium, with a feeling of constriction stretching round to loins and lasting 24 hours. Bowels had been regular. From beginning of illness had been confined to house, and mostly to bed.

On admission, chief complaint was of pain in hepatic region, vomiting, and weakness. Considerable emaciation and pallor; icteric tint of conjunctivæ, but no distinct jaundice and no anasarca. Appetite poor; thirst; tongue furred; bowels regular. Slight fulness in epigastrium; no ascites. Hepatic dulness extended in r. m. l. from sixth rib to 2 in. below costal arch, and in middle line to within two fingers' breadth of umbilicus; edge firm and rounded; surface smooth, firm, and tender. Spleen not perceptible below false ribs; but its dulness reached upwards to eighth interspace in axilla.

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[1] Dis. of Liver, Syd. Soc. ed. ii. 60.

During most of time he was in hospital, urine of normal quantity; sp. gr. 1010 to 1020; no albumen; no sugar; always traces of bile-pigment, and on one occasion crystals of leucin and tyrosin found after evaporation. Towards close it was scanty, high-coloured, and deposited much lithates. Pulse varied from 80 to 100. On some days temp. normal, but as a rule pyrexia, temp. varying from 99·5° to 101·5°. No periodicity in rise of temperature. Occasional perspiration, but not profuse, and never rigors. Three weeks after admission patient had an attack of pleuropneumonia in lower half of left lung, which subsided in a fortnight, and during which temp. rose to 103°. Pain and tenderness in hepatic region persisted, varying in intensity, but never very severe. Vomiting also occurred at times, and vomited matters were occasionally streaked with blood. Three weeks after admission diarrhœa set in; it was easily checked, but recurred at intervals; on two occasions stools contained a little blood; no piles. Moderate epistaxis about same time as hæmorrhage from bowels. Within a month of admission there was increased bulging of liver above umbilicus, and hepatic dulness extended about an inch higher up into chest. On two occasions an aspirator was passed deeply into different portions of liver without result. Epigastric veins became more prominent, but at no time was there ascites. Towards close diarrhœa became more frequent and obstinate, and at last was uncontrollable by remedies. Jaundice increased, but never intense; stools pale. Patient died exhausted six months after admission into hospital.

Autopsy.—Liver much enlarged, extending 4½ in. below ensiform cartilage, and 2 in. below ribs in r. m. l., and upwards as high as fourth intercostal space in front; weight 85 oz.; outer surface slightly granular. Hepatic tissue much indurated, at some places white, glistening, and replaced by fibrous tissue, at others showing a great increase of fibrous tissue between the lobules. Branches of portal vein dilated. Bile entered duodenum freely. Spleen enlarged and indurated. Stomach and intestines much congested. Lungs emphysematous, with some old calcareous nodules. Other organs healthy.

CASE CLXXIX.—*Interstitial Hepatitis resulting from Chill, and ending in Cirrhotic Contraction, in a Child aged* 12.

On Sept. 22, 1876, I was consulted respecting Miss Helen F., aged 12, and I received following particulars of her case from her mother and from Dr. Lewis Mackenzie of Tiverton, under whose care she had been. Gout on both sides of family. From infancy patient's liver had been sluggish. For two or three weeks at a time motions would be white and skin slightly yellow. At age of six, after bathing in sea, had been seized with severe abdominal pain, lasting twelve hours, and making her call out. For three weeks after had been poorly, with white stools &c. After this had usual health till autumn of 1874, when decided jaundice appeared for first time. It came on slowly, and was attended by some pain for about two days. Abdomen became very protuberant, with large veins coursing over it, and liver reached down almost to pubes. Kept bed for a day or two now and then. After three or four months general condition improved; but ever since had been thin; temper irritable, appetite capricious, bowels loose, urine very scanty, dark, and loaded with lithates, and complexion of a greenish hue. For weeks stools would be clay-coloured, without any trace of bile. Since March 1876 motions had often contained much mucus and bright red blood. Dr. M., who first saw her in March 1876, found spleen enlarged, but hepatic dulness diminished. At time of visit to me liver

not enlarged; spleen projected 2½ in. beyond ribs; slight jaundice, but no ascites; heart and lungs normal; no albuminuria; bowels loose, less blood; no vomiting; teeth decayed; no evidence of syphilis nor of taking wine or spirits; very thin and weak, but in last few weeks general condition had improved.

I wrote to Dr. M. as follows: 'Miss F.'s case is certainly an unusual one, but from the history I think there can be little doubt that she has some form of chronic interstitial hepatitis resulting in portal obstruction. The absence of the usual cause seems to negative true cirrhosis, nor is there any evidence that the hepatitis is syphilitic. I am inclined to attach importance to the attack of severe abdominal pain after bathing six years ago, as indicating the origin of the malady in a chill. But although the cause is obscure, the condition of the liver, I fancy, is very much this: thickening of capsule, with bands of fibrous tissue passing into interior and obliterating many of branches of portal vein.'

On Dec. 12, 1876, Miss F. died. For three weeks before death she was very ill. Symptoms were: diarrhœa with hæmorrhage; pulse 120, rising at last to 140; temp. at first 102·3°, falling at last to 95°; breath offensively sweet, like recently opened liver; delirium and maniacal excitement, and subsequently irregular breathing, stupor, and coma.

Autopsy by Dr. Mackenzie.—Liver small and rounded; weighed only 16 oz.; puckered and irregular on surface. Capsule thickened; tissue firm and dense, and pervaded everywhere by bands of fibrous tissue. Spleen enlarged. Lower portion of intestines much congested, and mucous membrane covered here and there with small extravasations. Mesenteric veins gorged with blood. Other organs normal.

Page 344. In Case CLXXX. it appears to me that the most probable explanation of the jaundice in the first instance was the presence of distomata in the bile-ducts, and that the development of the small amount of cancer found in the duodenum and portal lymphatics was contemporaneous with the rapid emaciation, ascites and severe pain, which set in five or six months before death. From what we know of other cases it is probable that in the early stage of the patient's illness there were a large number of these distomata in the liver, and very possibly it was distomata which the patient observed in the stools while at Malvern in June 1874. Distomata in the common bile-duct might have excited a local peritonitis in the portal fissure, leading ultimately to obliteration of the duct. It has been often observed that they cause great thickening and induration of the walls of the bile-ducts. The Fasciola hepatica, though very common in the livers of sheep and cattle, is very rare in the human liver. Possibly it may have often been overlooked. According to Cobbold, it has been met with in the human subject in only about twenty cases.[1]

CASE CLXXX.—*Jaundice from Distomata in Bile-ducts, followed by Cancer of Duodenum and Lymphatics in Portal Fissure—Ascites and Death.*

On May 8, 1874, I saw Mr. Charles B., aged 30, at the request of Dr. J. T. Williams of Barrow-in-Furness. He was of temperate habits, and had enjoyed

not enlarged; spleen projected 2½ in. beyond ribs; slight jaundice, but no ascites; heart and lungs normal; no albuminuria; bowels loose, less blood; no vomiting; teeth decayed; no evidence of syphilis nor of taking wine or spirits; very thin and weak, but in last few weeks general condition had improved.

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[1] Lect. on Practical Helminthology, 1872, p. 143.

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On May 8, 1874, I saw Mr. Charles B., aged 39, at the request of Dr. J. T. Williams of Barrow-in-Furness. He was of temperate habits, and had enjoyed

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On May 8, 1874, I saw Mr. Charles B., aged 39, at the request of Dr. J. T. Williams of Barrow-in-Furness. He was of temperate habits, and had enjoyed

excellent health until five months before, when he became jaundiced after overwork and worry. Jaundice came on gradually, with loss of appetite and lowness of spirits, but without pain or sickness. Skin had been intensely itchy and bowels irregular; motions light and urine dark. Since jaundice, but not before, had lost flesh at rate of a pound a week. No family history of malignant disease; father had been killed; mother had died at 73; one brother had died insane.

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[1] Lect. on Practical Helminthology, 1872, p. 143.

I noted that he was a small spare man, very nervous and excitable, and deeply jaundiced. Chief complaints were weakness, loss of appetite, and a coppery taste in mouth. Liver slightly enlarged; vertical dulness in r. m. l. measured 4¾ in. But what struck me most was a slight, but distinct, bulging forwards of costal cartilages to right of lower end of sternum; over this part was slight tenderness, but nothing like fluctuation.

The nature of the case was evidently obscure; but, in writing to Dr. W., I discussed fully the possibility of its being cancer, hydatid, or catarrhal jaundice. Cancer appeared excluded by patient's age, the absence of usual symptoms, and especially by fact that patient had been in good health and not losing flesh before jaundice appeared. On the whole I was inclined to think that, notwithstanding its duration, the case would turn out to be one of catarrhal jaundice, although the bulging referred to suggested the possibility of there being a hydatid.

I never saw patient again, but on several occasions I heard of him from Dr. W. On June 18, 1874, I heard that while at Malvern, about a fortnight before, he had felt something 'crack' in region of liver, and next morning stool had contained 'about a tablespoonful of little lumps of gum and small bladders. These Dr. W. imagined might be hydatids, but he had no opportunity of seeing any. Stools varied much, sometimes of colour and consistence of putty, at others almost natural in colour. Itchiness and bulging to right of sternum less. Lost about 7 lbs. more in flesh. Jaundice about the same. On Oct. 13, 1874, the report was as follows: For some weeks jaundice more intense, with increased irritability of skin. Stools very light and urine very dark. No change in bulging of right lower ribs. Appetite good, and more able to do his work. No pain nor uneasiness in region of liver at any time. On Feb. 3, 1876, the report was: 'In many respects better; has improved in strength, weight, and appetite, and, though usually very depressed in spirits, follows his employment as a clerk to the entire satisfaction of his employers. Motion snatural in colour and consistence. No irritation of skin, which is less yellow, but somewhat bronzed like that of Addison's disease. No appreciable bulging of right costal cartilages.' He remained much in same state and continued at his post until August 1876, when he noticed that he was beginning to increase in size around waist. The swelling, which was due to ascites, increased slowly and was attended by intense pain at lower end of sternum and rapid emaciation. Fæces were again light and often contained much mucus; urine loaded with bile. Towards end of December Dr. W. tapped abdomen and drew off nearly two gallons of yellow serous fluid; after this, pain was much relieved, but patient got weaker and thinner, and died on January 26, 1877. After death it was ascertained that Mr. B. had been particularly fond of uncooked shell-fish, especially whelks and mussels, but there was no evidence of his having eaten freshwater molluscs.

Autopsy.—Not more than 1½ pint of fluid in peritoneum. Dr. W. was good enough to forward to me liver, duodenum, and kidneys. Liver slightly granular on surface; substance dense, with increase of fibrous tissue between lobules. Common bile-duct and cystic duct completely obliterated by cicatricial contraction of fibrous tissue in portal fissure. Two or three lymphatic glands in portal fissure enlarged to about size of hazel-nuts and compressing trunk of portal vein. Gall-bladder greatly distended, containing fully 10 oz. of colourless, serous, flaky fluid. Bile-ducts in interior of liver moderately dilated; and one of them contained a fine specimen of Distoma (Fasciola hepatica);[1] ducts were carefully washed out, but only the one specimen could be found. Mucous membrane of duodenum, not far from orifice of bile-duct, contained a circular somewhat elevated plate of morbid deposit, about size of a shilling. This deposit was not ulcerated and did not extend into muscular coat; but both it and enlarged glands in portal fissure were ascertained by Dr. Greenfield to be cancerous. No evidence of cancer elsewhere in body. Left kidney expanded into a large cyst which ruptured in removal; nature of this cyst not ascertained.

[1] The specimen is in the Museum of St. Thomas's Hospital.

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Abbreviations: L. = Liver. J. = Jaundice. F.D.L. = Functional Derangements of Liver.

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